The Journal of
Thoracic Surgery

VOL. 21 JUNE, 1951 NO. 6

Original Communications

A CONSIDERATION OF SOME OF THE PROBLEMS IN CARDIOVASCULAR SURGERY

ALFRED BLALOCK, M.D.
BALTIMORE, MD.

DURING the last several decades there has been a progressive increase in deaths attributable to cardiovascular disease. It is estimated[1] that 44 per cent of all deaths in the United States in 1948 were due to disorders of the heart and vessels, and that there are at present nine million people in this country with some type of disease of the cardiovascular system. As the length of life increases, derangements of this system assume added importance. Hypertension, arteriosclerosis, and rheumatic fever make up the great bulk of cardiovascular problems, and, unfortunately, much remains to be learned about the etiology as well as the prevention and treatment of these diseases. It is to be hoped in the future that appropriate preventive measures will greatly reduce the mortality and morbidity of cardiovascular disease in general. Even if this hope is realized surgery will still have a role to play, particularly in the treatment of the complications of a mechanical nature.

Excluding peripheral vascular diseases such as arteriosclerosis, Raynaud's disease, Buerger's disease, and varicose veins, the major proportion of cardiovascular cases now amenable to surgery are of congenital or traumatic origin. Trauma, the cradle of surgery, will continue to contribute its quota, even if world conflicts should disappear. Preventive measures such as the possible elimination of rubella may result in a decrease in the incidence of congenital heart disease, but the chances are that congenital disorders will be of fairly frequent occurrence for centuries to come.

It would be unfair to the field of cardiovascular surgery to judge its future potentialities on the basis of present accomplishments, since this field is as much an advancing one as is that of preventive medicine and that of nonoperative therapy. For example, a fundamental advance which would solve the problem of homotransplantation of tissue would open up great potentialities in cardiovascular surgery.

This lecture is not intended to be an all-inclusive consideration of the subject. For example, peripheral vascular disease and portal hypertension will

From the Department of Surgery of The Johns Hopkins University and Hospital.
Presidential Address, Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, April 16, 17 and 18, 1951.

not be discussed. Greater attention will be devoted to congenital heart disease because of the recent advances in this field. I have arbitrarily divided the disorders to be considered into three groups: (1) those in the treatment of which surgery may bring about good or excellent results, (2) those in which surgical treatment may cause moderate improvement, and (3) those in which surgical treatment is of doubtful value or is as yet undeveloped. There is a justifiable difference of opinion as to the group in which some of the disorders should be placed. The main emphasis will be on unsolved problems rather than on accomplished facts.

The conditions will be discussed in the following order:

I. Disorders in the Treatment of which Surgery may Accomplish Good or Excellent Results
   - A. Acquired Lesions
     1. Constrictive Pericarditis
     2. Wounds of the Heart
     3. Systemic Arteriovenous Fistula
     4. Arterial Aneurysm
     5. Mitral Stenosis
   - B. Congenital Lesions
     6. Patent Ductus Arteriosus
     7. Coarctation of the Aorta
     8. Pulmonary Stenosis and Atresia
     9. Pulmonary Arteriovenous Fistula
     10. Anomalies of the Aortic Arch

II. Disorders in the Treatment of which Surgery may Bring about Moderate Improvement
   - A. Acquired Lesions
     11. Essential Hypertension
   - B. Congenital Lesions
     12. Transposition of the Aorta and Pulmonary Artery
     13. Anomalies of Venous Return

III. Disorders in the Treatment of which Surgery is of Doubtful Value or Suitable Methods are not yet Fully Developed
   - A. Acquired Lesions
     14. Coronary Arterial Disease
     15. Aortic Valvular Stenosis
     16. Insufficiency of Heart Valves
     17. Intracardiac Tumors
   - B. Congenital Lesions
     18. Anomalous Origin of Left Coronary Artery
     19. Auricular Septal Defects.
     20. The Lutembacher Syndrome
     21. Ventricular Septal Defects
     22. The Eisenmenger Complex
     23. Pulmonary Hypertension

I. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY ACCOMPLISH GOOD OR EXCELLENT RESULTS

A. Acquired Lesions.—

1. Constrictive Pericarditis: This is now a well-recognized clinical entity which usually presents few difficulties in diagnosis. It is often impossible to determine the cause. In most such cases it is likely that the tubercle bacillus

is the offender. The use of chemotherapeutic and antibiotic agents in the prevention and control of infections will probably result in a reduction in the incidence of constrictive pericarditis.

The fundamental surgical problem remains the same as that enunciated by Delorme,[2] namely, the removal of the scar which is constricting the heart and in some cases the great blood vessels. It is necessary to remove the scar overlying the epicardium as well as that which lines the pericardium.

Prior to the discovery of streptomycin and similar antibiotic agents, it was held that patients with tuberculous constrictive pericarditis should not be operated upon until the process became inactive. One of the reasons for this viewpoint was that operation subjected the patient to the dangers of a systemic as well as a localized spread of the disease. A second reason given was that it is extremely difficult to remove the shaggy exudate that is present in the subacute stage. It is the opinion of Holman[3] that antibiotic therapy may now allow one to operate with safety during the earlier stages of the disease. Certainly these agents will lessen the chances of spread of the disease, and possibly it will not be necessary to remove the tuberculous granulation tissue covering the surface of the heart. No one has yet had sufficient experience with the combined use of surgery and antibiotic therapy in the treatment of active tuberculous pericarditis to give an authoritative answer.

The types of incision that may be employed in decortication of the heart include a parasternal one with resection of costal cartilages, a median sternotomy, and an intercostal transpleural approach. At the present time I prefer the latter incision for it gives more adequate exposure than the others. A long left anterolateral incision is made overlying the fourth interspace, and the fourth and fifth costal cartilages are divided at their sternal attachments. Through this incision one can expose and decorticate both the left and the right side of the heart, including the superior and inferior vena cava. It makes little difference if the right pleura is torn, provided one recognizes the condition and maintains inflation of the lung. Recent experience has shown that thorough decortication usually causes a prompt decline in the venous pressure and general improvement of the patient. Our results with this incision are much better than those obtained when a parasternal extrapleural incision was used.

2. Wounds of the Heart: As has been true of developments in many fields of surgery, the treatment of wounds of the heart and blood vessels was the springboard for further advances in cardiovascular disorders. It is generally agreed that wounds of the heart should be operated upon if there is continuing bleeding to the outside or into the pleural cavity. Fortunately, various advances in surgery, including improvements in anesthesia, have decreased the mortality rate when operation is necessary. There is some difference of opinion how the patient with uncomplicated acute cardiac tamponade without continuing bleeding should be treated. Dr. Ravitch and I[4] are of the opinion that open operation should be deferred until the effects of at least one pericardial aspiration have been determined. It has been our experience that the majority of patients will respond favorably to one or more aspirations, that

bleeding into the pericardial cavity will not recur, and that an open operation will not be necessary. Elkin and Campbell[5] have recently reported on the effectiveness of aspiration in the treatment of cardiac tamponade.

3. Systemic Arteriovenous Fistula: The majority of such fistulae are traumatic in origin, and hence are considered in this group. The diagnosis and the treatment of traumatic systemic peripheral arteriovenous fistula are clear-cut. The mortality in the many patients operated upon during World War II was amazingly low. It is generally agreed that the best treatment consists of eradication of the fistula with repair of the openings in the artery and vein, and that the second method of choice is quadruple ligation of the artery and vein with excision of the fistula provided the continuity of the artery can be interrupted without the loss of distal parts. There is some difference of opinion as to the choice of time of operation. If the artery involved is one which can be spared if necessary, one may operate as soon as the fistula is discovered. If sacrifice of the involved artery would subject its part to the danger of gangrene, an interval of a few weeks should be allowed to elapse following the injury in order to permit the further development of collaterals.

Most of the congenital peripheral arteriovenous fistulae involve bone, and here the problem of treatment may be an exceedingly difficult one. If the bony involvement is limited,[6] one may excise the segment with or without replacement with a graft. Unfortunately, however, the lesions are usually extensive, and multiple amputation may be necessary.

The subject of intracranial arteriovenous fistulae is a difficult one with many unsolved problems and will not be discussed.

4. Arterial Aneurysm: With better control of syphilis, the incidence of aneurysms should lessen. On the other hand, there will probably be an increase in those due to arteriosclerosis unless preventive and therapeutic measures decrease the incidence of this disease. Unfortunately, the arteriosclerotic process is usually not limited to the aneurysm itself, and this fact renders it important in surgical treatment to interfere as little as possible with the blood supply of parts distal to the aneurysm. The Matas endoaneurysmorrhaphy, the wiring procedure as used by Finney and others and further advanced by Blakemore, and the placing of irritating cellophane around the sac have in common the advantage that they need not interfere unduly with the collateral arterial pathways around the aneurysm. This is particularly true of the wiring procedure. Blakemore[7] now treats some aneurysms by constricting the artery proximal and distal to the aneurysm with polythene tape, thereby reducing the pulse pressure, and by heating wire introduced into the sac. The relief of pain and the abolition of respiratory difficulty that may follow the wiring of a thoracic aneurysm is a dramatic accomplishment. If the results that are being obtained by Gross[8] and others on the homotransplantation of aortic grafts continue to be encouraging, it is likely that the procedure can be used in conjunction with excision of the aneurysmal sac when the neighboring segments of aorta are not grossly diseased. Lam[9] has inserted an

aortic graft without excision of the sac. The development and use of improved plastic materials may be helpful in the treatment of aneurysms.

5. Mitral Stenosis: It may seem unduly optimistic to place this disorder in this group, but I do so because of the surprisingly good early results that are being obtained at present. Most cases of mitral stenosis are due to rheumatic fever, and it is probable that great advances will be made in the prevention or treatment of rheumatic infections. Even without further advances, it is likely that the use of antibiotic and other agents which are available will reduce the repeated attacks of infection and thereby lessen the incidence of mitral stenosis. On the other hand, many attacks of acute rheumatic fever are unsuspected or undiagnosed and the later development of some chronic disorder may give the first unmistakable clue to an earlier attack of an acute infection. For this reason it is likely that there will continue to be a moderate number of cases of mitral stenosis despite preventive and therapeutic advances.

Following the discouraging attempts[10, 11] which were made to treat mitral stenosis by surgical methods in the third decade of the present century, it is largely to the credit of the late Horace Smithy,[12] Bailey,[13], and Harken[14] that the problem was revived several years ago. In view of the present encouraging results, in some of which the procedure consists of dilation of the stenotic valve with the finger, it is difficult to understand why the successful use of this method by Souttar[15] in 1925 did not stimulate greater interest at the time. Whereas Cutler and his associates believed that a segment of the stenotic valve should be removed, and a similar view was held by Smithy, both Bailey and Harken are of the opinion that dilation of the valve and division or tearing of the commissures produce an opening of adequate size and that mitral insufficiency does not result. Both Bailey and Harken prefer an approach through the left auricular appendage as was advocated by Allen and Graham, and there is no doubt that this is preferable to that through the left ventricle. Bailey employs a knife which is attached to the index finger for division of the commissure region. Harken attempts to fracture or tear the commissure region with the index finger, and if this fails he divides it with a knife. It is our experience that in most of the cases one or both of the commissure regions can be torn with the index finger and that the use of a knife is generally unnecessary. Bailey does not favor the making of much pressure with the finger because of fear of tearing one of the valve leaflets. We have not as yet had the unfortunate experience of tearing one of these. It should be emphasized that it is not always possible to tear the commissure region and that one should have a proper cutting instrument available.

The early results that are being obtained in the treatment of mitral stenosis by direct attack on the valve are surprisingly good. The subjective improvement of the patient is usually greater than the alteration in the measurable dynamics of the circulation. It is too early as yet to know what the long term results will be, but even the improvements of the duration that have been recorded to date justify the operation. Subsequent observations may show that there is a gradual reformation of the stenosis. If such is the case, it may

be advisable to remove a segment of the valve, the plan of attack which was used by Cutler, Graham, Smithy and their associates. Since there are still unsolved problems, it is important that efforts to produce mitral stenosis in experimental animals be continued.

In addition to recent progress in the direct attack on the stenotic mitral valve, efforts have been made to prevent bouts of pulmonary edema in this condition by the anastomosis of systemic and pulmonary veins as performed by Sweet[16] or the making of an interauricular defect as advised by Harken.[17] It is obvious that these shunting procedures should not be used if there is a very tight stenosis, indicated by small left ventricular output and inability to increase it with exercise, for a further reduction in output caused by the shunt will not be tolerated. There are probably some cases in which some such procedure will be helpful, but in general it would appear that the surgical treatment of mitral stenosis should consist of a direct attack on the stenotic area itself.

Much probably remains to be learned about the choice of patients in whom a direct attack on the mitral valve is indicated. According to Andrus, with whom I have been associated in this work, the indications for a direct attack on the stenotic mitral valve are as follows: Symptoms and signs of pulmonary engorgement—dyspnea and cough on exertion, paroxysmal dyspnea, pulmonary edema, or frank hemoptysis. The most promising cases are those without enlargement of the left ventricle or prominent enlargement of the left auricle; usually these are signs that mitral insufficiency contributes significantly to the patient's disability. In general, there should be predominantly a stenosis, although a small degree of insufficiency is not a contraindication to commissurotomy. If one uses fairly rigid criteria in the selection of patients, the mortality associated with operation is fairly low. One of the twenty-five patients upon whom we performed a commissurotomy died postoperatively. An additional patient who was found at exploratory thoracotomy to have an obliterated auricular appendage, no commissurotomy being performed, succumbed subsequently from a cerebral embolus.

B. Congenital Lesions.—

6. Patent Ductus Arteriosus: Although a procedure for the correction of patent ductus arteriosus had been suggested many years previously by Munro[18] and had been attempted by Strieder,[19] it is to the everlasting credit of Gross[20] that he was the first to close a patent ductus successfully. The typical case is not difficult to diagnose, and technical procedures are available for obtaining a permanent closure of the ductus with a low mortality. In view of these facts, and because of the dangers of allowing a ductus to remain patent, it is my belief that operation is indicated for all children beyond two or three years of age who have an obviously patent ductus arteriosus. However, it is often difficult to be certain of the diagnosis in infants. Furthermore, Dammann[21] and Keith[22] have recently called attention to the fact that some patients with a very large patent ductus and associated pulmonary hypertension

may not have a typical machinery-like murmur. In some of these patients the presence of a large patent ductus has been confirmed by thoracotomy. There is some difference of opinion whether one should divide the ductus and close the two ends as preferred by Gross[23] and others, or whether one should close the undivided ductus by the use of multiple suture ligatures placed as far apart as possible. My associates and I prefer the multiple suture technique[24] because it seems to us that it is easier and safer. The desideratum is permanent closure of the ductus, and we are aware of only one instance of recanalization in the approximately 300 patients in whom we have used this method.[25] The exception was a child of five who had a ductus which measured 16 mm. in diameter.

Whereas the operation for closure of a patent ductus is not a very dangerous one in most children and young adults, the procedure becomes more difficult and dangerous in the older age groups. Pulmonary hypertension with disease of the walls of the pulmonary artery and ductus increases the likelihood of uncontrollable bleeding from these structures. In the older patients with an enlarged heart and pulmonary hypertension we use a long posterolateral incision instead of the anterolateral incision which is preferred in the less complicated cases. In the occasional patient with extreme pulmonary hypertension in association with a patent ductus there is some flow of blood from the pulmonary artery to the aorta. This reverse flow of blood is particularly apt to occur if the patent ductus is attached to the aorta distal to a coarctation of the aorta. Closure of the ductus is especially dangerous in patients with severe pulmonary hypertension and a reversal of flow through the ductus. In addition to the hazards of hemorrhage, there is the possibility that closure of the ductus will result in a sudden increase in the diastolic volume of the right ventricle and undue distention of that chamber.

Now that the diagnosis of the patent ductus can be made in most instances with accuracy and the operative technique is perfected, it is to be hoped that the condition will be recognized and treated in childhood and that in the future fewer cases will be encountered in adults.

An aortic septal defect providing a communication between the aorta and the pulmonary artery may give signs and symptoms similar to those of the usual patent ductus. A small aortic septal defect may be closed by existing techniques, but the large one extending to the base of the heart presents problems that have not been satisfactorily solved.

7. Coarctation of the Aorta: Fortunately there is usually no difficulty in the diagnosis of coarctation of the aorta. If there is any reason to suspect that the coarctation is at an unusual site, angiocardiography or aortography should be used in demonstrating the lesion. It is not necessary to use these radiological methods in the majority of cases.

The proper treatment of the adult type of coarctation, the common form in which the constricted segment is short, is essentially that which was first employed successfully by Crafoord,[26] and consists of excision of the stenotic or atretic segment and an end-to-end anastomosis of the aorta. It is desirable

to preserve the dilated intercostal arteries. There is some difference of opinion as to the type of suture that should be used in making the anastomosis. We usually employ a continuous everting suture of silk which is interrupted at several points. An overhand noneverting suture also gives satisfactory results. Interrupted sutures probably cause the least disturbance of growth of the anastomotic site and should be used in young children. Several recent experimental studies indicate that the size of the lumen at the site of the anastomosis increases somewhat with the growth of the subject even though a continuous suture is used. This brings up the question as to the age period in which the operation should be performed. I think the ideal period is from six to fifteen years of age. Complications in connection with coarctation rarely occur before the age of six, although in one of our patients hemiplegia developed at the age of two years. The aorta of a child of six is usually of sufficient size to provide a good lumen following anastomosis even though subsequent growth of this site may not be great. The employment of interrupted sutures will increase the likelihood of growth. I have no strong convictions as to the upper age limit at which operation should be advised. We have operated upon a number of patients in their thirties and one in the early forties with good results. On the other hand, I am inclined to think that a patient past forty who has no serious symptoms or complications save for the hypertension accompanying the coarctation should not be operated upon.

The treatment of the infantile type of coarctation in which the constricted segment is long presents a more difficult problem. The same may be said for the occasional case of the adult type when the proximal or distal segment is hypoplastic for a short distance. I think a good result will be obtained in most cases by the use of the left subclavian artery[27] to bridge the narrowed segment if the procedure is performed properly. This means that the constricted segment should be excised in toto, and the proximal end of the aortic segment should be closed with an arterial suture; the left subclavian artery should be cut across proximal to its initial branching, and should then be rotated downwards in such manner that there is no sharp twisting or angulation, and its end should be anastomosed to the distal end of the aorta. If one performs the operation without removing the constricted aortic segment and closing the proximal end of the aorta and without proper rotation of the subclavian, a good straight channel for the flow of blood is not obtained. Usually the subclavian artery is almost as large as the distal aorta, and there is little difficulty in performing a good union. The other alternative is the employment of a homograft of aorta as used by Gross[8] to replace the constricted segment of the aorta. The early results in the experience of Gross have been amazingly good. It is too early as yet to know how the homograft will weather the passing of time. The chances are that it is replaced by the recipient's own tissues. Disadvantages of this procedure are that it is difficult to obtain proper grafts and that two arterial anastomoses have to be performed. The severe constriction of the aorta that is present in the infantile type of coarctation should usually be discovered in childhood, and the use of

either the transposed subclavian artery as a substitute for the segment or the employment of homografts should give fair results. Unfortunately, the stenosis may be proximal to the origin of the left subclavian artery and may be associated with other congenital defects.

The question remains unsettled why some patients who are operated upon for the relief of coarctation do not have the desired decline in blood pressure. Fortunately, this is the exception rather than the rule. Among the possibilities is the fact that even after operation the aortic lumen in some patients is still not as large as it should be. This is particularly likely to be the case when the lumen of the proximal aortic segment is smaller than normal. Another possibility, if the kidney is involved in this type of hypertension, is that renal changes may persist after operation. Still another possibility is that irreversible alterations in the arterioles may have occurred in connection with the hypertension. I think that the number of disappointing results will decrease as additional experience is gained by the performance of larger and more perfect anastomoses. We are fairly well pleased with the results of operations on 104 patients with coarctation.

8. Pulmonary Stenosis and Atresia: The most frequent example of this type is the typical tetralogy of Fallot.[28] Other types of malformations which may be associated with pulmonary stenosis or atresia include tricuspid atresia, single ventricle, pseudotruncus arteriosus, dextrocardia or dextrorotation of the heart, situs inversus viscerum with levorotation, and valvular pulmonary stenosis with intact ventricular septum.

The typical case of *tetralogy of Fallot* presents few difficulties in diagnosis. There is need for more accurate appraisal of the degree of overriding of the aorta, the length of the infundibular stenosis, and the size of the infundibular chamber since this information may alter treatment, as will be discussed later. Patients with excessive dextroposition of the aorta may not do well following the creation of an artificial ductus. With the increase in the blood flow to the lungs there will obviously be an increase in the amount of blood returning to the left side of the heart. Since in cases of extreme dextroposition the aorta receives blood mainly from the right ventricle unless the interventricular defect is large, the left ventricle may be unable to empty itself properly and pulmonary engorgement and edema may develop. One possible means whereby the degree of overriding might be determined more accurately is by the injection of radiopaque material through a catheter placed in the right ventricle and the taking of repeated roentgenograms. The possible value of this procedure will be determined only by multiple trials. If an extreme degree of overriding were proved to be present, one would certainly not wish to create a large artificial ductus. Perhaps some day a safe means will be available for increasing the size of the interventricular defect. This could be done at the same time that the ductus is made.

It is largely as a result of the observations of Brock[29] that the efforts toward a direct attack on infundibular stenosis rather than the creation of an artificial ductus have been stimulated. The direct attack is preferable to the

anastomotic procedure, on theoretical grounds at least, because it reduces the shunt through the interventricular defect at the same time that it increases the pulmonary flow, and it does not add another anomaly to those already present.

Some idea as to the size of the infundibular chamber and the length of the stenosis may be obtained by angiocardiography. The information might be more exact if the diodrast were injected through a catheter introduced into the right ventricle. In addition to the information gained in preoperative studies, one can obtain some idea as to the size of the infundibular chamber by exposing this region at the time of operation. There are certainly many cases of the tetralogy of Fallot in which there is a long infundibular stenosis and in which a direct attack on the stenosis is not feasible. There are probably a moderate number of cases with a short diaphragm-like zone of stenosis and a large infundibular chamber in which a direct attack is possible. As indicated previously, there is need for more accurate means for determining the exact condition of the lesion. In some patients with a short infundibular stenosis it is not difficult to introduce dilating and cutting instruments through an incision in the right ventricular wall and through the stenotic area, as we have done in a few cases following the lead of Brock. It is much more difficult and dangerous to excise part of the stenotic area as performed by Brock[30] and Glover,[31] and others. Technical improvements are needed in this respect. Furthermore, the question remains unsolved whether such a stenotic area in the heart muscle which is treated by dilation and incision or excision will subsequently reform. It is my opinion that improvements in accuracy of anatomic diagnosis and in the technique of intracardiac procedures will result in an increasing number of direct attacks on infundibular stenosis. At the same time Dr. Taussig and I believe that the majority of these patients will have to be treated by the creation of an artificial ductus. Certainly this latter procedure rather than a direct attack offers hope of improving the patient with pulmonary atresia. In this connection it should be mentioned that simple exploratory thoracotomy with the subsequent formation of adhesions and the resulting additional collateral blood supply to the lungs seems to improve the condition of some patients in whom the pulmonary artery is not of suitable size for an anastomosis. The number of collateral channels is probably increased by removing the parietal pleura as recommended by Barrett.[32]

The techniques that are available for the creation of an artificial ductus are well developed, and the results are amazingly good in the typical tetralogy of Fallot. Dr. Taussig[33] is of the opinion that the occasional patient who does not do well following a satisfactory anastomosis does not have an uncomplicated tetralogy of Fallot. We perform an end-to-side, an end-to-end or a side-to-side anastomosis between a systemic and a pulmonary artery, the choice depending upon the age and size of the patient and the observations at the time of operation. This choice has been considered in detail in a previous publication.[34] In the 20 odd per cent of patients in whom the aorta descends on the right, the incision is almost invariably made on the left, and an anastomosis performed between the left subclavian and the left pulmonary artery.

In patients in whom the aorta descends on the left, the incision is usually made on the left in infants, in adolescents, and in adults, and it is usually made on the right in children between the ages of two and twelve years. In patients in whom there is considerable doubt as to the diagnosis, for example when valvular pulmonic stenosis is suspected, the incision is made on the left in order that one may examine the structures at the base of the heart. When the incision is made on the left in the presence of a left descending aorta, the left subclavian artery is used for the anastomosis if it is of adequate size and length; otherwise the aortic side-to-side anastomosis as devised by Potts[35] is employed. We do not hesitate to use an end-to-end anastomosis between the subclavian artery and the divided distal end of one of the pulmonary arteries if the pulmonary artery is unusually small or if the condition of the patient is poor and one wishes to perform the procedure speedily and without traction on the mediastinum. Dr. Taussig and I consider the optimal time for operation to be between the ages of five and ten years. A second operation at a later period is less apt to be necessary if the child has reached the age of five or more before the initial operation is performed. Unfortunately, the condition of many infants and young children is such that earlier operation is required. The general philosophy of Dr. Taussig's cardiac clinic is, "The sicker the sooner."

The results of the creation of an artificial ductus in the treatment of pulmonary stenosis or atresia are not so good when there are associated malformations such as tricuspid atresia, single ventricle, dextrocardia or dextrorotation, or situs inversus with levorotation, as they are in the typical uncomplicated tetralogy. Tricuspid atresia is usually associated with a diminutive or absent right ventricle and some abnormality of the pulmonary orifice. Some patients with this condition are improved by the creation of an artificial ductus. Others do poorly, due in part at least to a resulting increase in strain on the left ventricle in the absence of an interventricular defect. Patients with tricuspid atresia and with only a small opening between the two auricles may be improved by the creation of a large interauricular defect. If one employs the method which Hanlon and I[36] devised for this purpose, one has to be very careful during the procedure not to occlude entirely the existing communication between the two auricles for more than a few seconds. Patients with a single ventricle respond very poorly to the creation of an artificial ductus, and the operation should not be advised if the diagnosis can be made and if the patient is getting along moderately well. Patients with dextrocardia, or dextrorotation of the heart, or situs inversus viscerum with levocardia are likely to have a variety of malformations and are likely to respond unfavorably to the creation of an artificial ductus.[37]

Valvular pulmonary stenosis with or without a communication between the two auricles and with an intact ventricular septum occurs approximately 10 per cent as often as the tetralogy of Fallot. The work of Brock,[29] of Sellors,[38] and of others has added materially to the diagnosis and treatment of this condition. Cyanosis is present in about 70 per cent of the cases and is due to a right to left

shunt through a patent foramen ovale or an auricular septal defect. The pooling of diodrast as observed on angiocardiography in the pulmonary artery distal to the valvular stenosis is an important clue in diagnosis. The pressure in the right ventricle is significantly elevated and may exceed that in the left ventricle. Bing recorded a right ventricular pressure of 223 mm. Hg in one of our patients. The conditions observed at the time of operation are characteristic. There is definite right ventricular hypertrophy, usually a poststenotic dilatation of the pulmonary artery, a jet of blood can be felt striking the wall of the pulmonary artery, and usually the dome-shaped pulmonary valve can be palpated. Patients with valvular pulmonary stenosis are not improved by the creation of an artificial ductus. It is true that cyanosis if present is lessened, but these patients subsequently have right-sided heart failure.

We have followed essentially the technique of Brock in treating valvular pulmonary stenosis.[39] The approach is through an incision in the right ventricle, and the valve is cut and dilated. The Brock flat valvulotome with a probelike end and two sharp cutting edges is a very satisfactory instrument. Even so, the umbrellalike instruments recently developed by Potts present some advantages. In general we have been greatly pleased with the results obtained but a number of questions are as yet unanswered. Among these are the following: (1) Why is it that the blood pressure usually declines and the heart may fail shortly after opening the chest and exposing the heart? (2) Why is it that the pressure in the right ventricle may not fall to normal following division and dilation of the valve and in the presence of definite improvement in the condition of the patient? (3) Should operation be advised prior to the development of great incapacity and of right ventricular hypertrophy and dilatation? (4) Will extreme right ventricular hypertrophy, once developed, ever disappear entirely following operation? (5) Should the stenotic valve simply be incised and dilated as is done at present, or should a segment of the valve be excised? (6) When the valve is incised and dilated, do the incisions in the leaflets remain open or do they partially grow back together?

A longer postoperative follow-up of the patients and a repetition of the studies will probable supply an answer to some of the above questions. It is known that patients with great enlargement of the heart are those in whom difficulty is most likely to be encountered during the operation, and I believe that patients should be operated upon in advance of this enlargement if the condition is diagnosed. It has been observed in some of our patients that the size of the right side of the heart has decreased postoperatively, but this may be due to a disappearance of the dilatation rather than to a regression of the hypertrophy. The finding of Dr. Bing[40] that the pressure in the right ventricle usually does not fall to normal in the first few months after operation may be explained on the basis of inadequate division and dilation of the valve or possibly on the basis of partial obstruction at the pulmonary orifice by the greatly hypertrophied right ventricular muscle. I have already commented on the fact that patients show decided improvement postoperatively despite the persistence of some right ventricular hypertension. The valve in congenital

pulmonic stenosis is only moderately thickened, and I believe that incision and dilation will result in a permanent increase in the size of the opening. It appears likely that endothelium grows over the incised area and that the opening remains patent. If such should prove not to be the case, it will be advisable to remove part of the valve as has been performed by Varco.[41] No ill effects attributable to pulmonary insufficiency have been observed in any of our patients subjected to pulmonary valvulotomy, and it would seem that patients should withstand removal of part of the valve without ill effects.

9. Pulmonary Arteriovenous Fistula: This condition is being diagnosed with increased frequency and should be suspected in cyanotic patients in whom there is no evidence of disease of the heart itself. In doubtful cases, angiocardiography is helpful in diagnosis. Evidence of abnormal communications between pulmonary arteries and veins may be late in developing. Unfortunately, the communications are usually multiple and parts of both lungs may be affected. Treatment consists of excision of the diseased areas; in some instances a wedge-shaped resection will suffice. If a lobe is extensively involved it should be removed. One should not, however, remove an entire lung unless there are no normal segments present for fear that evidence of abnormal arteriovenous communications will subsequently appear in the remaining lung. One of the most dramatic results which I have witnessed in surgery followed the removal of a lobe of the lung containing multiple arteriovenous communications from a 28-year-old woman. Total incapacity was replaced by perfect health. The saturation of the arterial blood with oxygen is now normal and there has been a gain of 36 pounds in weight.

10. Anomalies of the Aortic Arch: Anomalies of the aortic arch and its major branches occur frequently in patients with congenital heart disease, but only a few of these in themselves cause any difficulty. A small proportion of these anomalies interfere with breathing or swallowing or both due to pressure on the trachea and esophagus. This difficulty is particularly likely to be present if there is a bifid aorta, although the same symptoms may be caused occasionally by a retro-esophageal artery such as an aberrant subclavian. Neuhauser[42] has made important contributions to the diagnosis of this condition. Gross[43] and others have shown that the treatment consists of the division of the offending blood vessel. In the case of the bifid aorta, the smaller of the two arches should be divided and the two ends closed. The most interesting patient with a bifid aorta in our experience is a boy who had tetralogy of Fallot as well. Treatment consisted of division of the smaller arch and an anastomosis of its distal end to the left pulmonary artery. In general, surgical treatment of anomalies of the arch is satisfactory.

II. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY BRING ABOUT MODERATE IMPROVEMENT

A. Acquired Lesions.—

11. Essential Hypertension: Whereas there is a high percentage of good results in the treatment of hypertension associated with coaretation of the

aorta, pheochromocytoma and unilateral renal disease, the outlook in essential hypertension is less promising. It is true that some patients with essential hypertension obtain an excellent result after sympathectomy, and one may therefore criticize the placing of this disorder in this group. I do so because it would seem, in view of follow-up studies, that the ultimate treatment of hypertension will not be by surgical sympathectomy. A recent report[44] of a thirteen-year period showed that 21 per cent of the patients were improved, but only 8 per cent had a normal blood pressure five years after their operation. At the same time, if medical measures fail, sympathectomy is the best that we have to offer at present. It is possible that when more is known about the cause of essential hypertension, some operation on the central nervous system will be helpful. It is more likely that ultimately a successful nonoperative form of treatment will be found.

B. Congenital Lesions.—

12. Transposition of the Aorta and Pulmonary Artery: This type of congenital malformation is rather common if one includes those instances in which death occurs shortly after birth. Since the aorta and the pulmonary artery are transposed but the systemic and pulmonary veins occupy their normal positions, there has to be some type of communication between the two sides of the heart if there is to be any chance of survival. Usually there is an interventricular defect, an interauricular defect, a patent ductus, or various combinations of these lesions.

Adequate means are available for the diagnosis of transposition of the great arteries. The problem of surgical therapy is an exceedingly difficult one and is far from solution. Not only is there an inadequate mixing of blood of the two circulations but there is also severe pulmonary hypertension. We have been fairly well pleased with the results that have been obtained in some of our patients in whom a large interauricular defect was created.[45] In some instances this was accompanied by the making of an artificial ductus of the end-to-end type. The results have been fair in children and very poor in infants. Some of our best results have been obtained in patients who had pulmonic stenosis as well as transposition of the great arteries and also in those in whom the transposition was of the Taussig-Bing type[46] in which the pulmonary artery overrides an interventricular defect. A smaller percentage of those children with a typical transposition have been definitely improved.

The problem of therapy has been made more difficult by inability to reproduce transposition in toto in the experimental animal. Actually Hanlon and I have produced by a multiple stage procedure what amounts to a total transposition, but the right ventricle was unable to carry on the systemic circulation effectively. These experiments are being continued.

It appears on first thought that one's efforts should be directed toward the development of methods which would allow one to cut across the pulmonary artery and aorta of the patient with transposition and to transpose or correct the position of these vessels, using a suture anastomosis. It is possible that such a procedure may be carried out after perfection of the artificial

heart and lung. Even if this proves to be feasible ultimately, there is still the problem that the coronary arteries could not be transposed and that they would continue to receive venous blood from the right ventricle. Almost certainly the oxygen content of this blood would be higher than before the positions of the aorta and pulmonary artery were corrected, and it is possible that it would suffice for the nourishment of the myocardium. On the other hand, it is known that infants with an anomalous origin of coronary arteries from the pulmonary artery usually do poorly. However, the pulmonary arterial pressure in these patients is low whereas it is high in those with transposition. Even if it becomes technically feasible to correct the position of the aorta and the pulmonary artery except for their orifices, and even if the myocardium should receive adequate oxygen from venous blood, there still remains the problem of the pulmonary hypertension. It is possible but not at all certain that it would disappear after correction of the position of the great arteries.

Since one could not alter the position of the coronary arteries even if one were able to correct the positions of the aorta and the pulmonary artery, perhaps the ideal would be the development of methods whereby the systemic and pulmonary veins could be transposed, leaving the aorta and the pulmonary artery in their transposed positions. If total transposition of the venous return could be accomplished successfully it would probably result in great improvement in the condition of the patient. The coronary arteries would receive arterial blood, and one's greatest concern would be with what would happen to the pulmonary hypertension. Unfortunately, an experimental complete transposition of the systemic and pulmonary veins is difficult if not impossible with existing techniques. Brown and I[47] have accomplished in the experimental animal a transposition of the pulmonary venous return, but this required the removal of most of the left lung and the making of a large interauricular defect. Anastomoses of veins to the auricular appendage or auricle are very likely to become occluded. Anatomic relationships add to the difficulties of producing complete transposition of veins, such as, for example, the fact that the inferior vena cava is some distance from the left auricular appendage. Transposition of some of the pulmonary veins can be produced without too much difficulty, and we used this method to a limited extent in our early efforts to treat transposition in patients. Unfortunately patients with transposition of the great arteries tolerate a partial alteration in venous return very poorly since it causes an imbalance between the two sides of the heart. The situation is totally different from that existing in the normal heart where the blood leaves from one side and returns to the other.

After this lengthy discussion it should be repeated that our best results in the treatment of transposition of the aorta and pulmonary artery have followed the creation of a large interauricular defect. We are encouraged but are not satisfied with the results, and better methods should be devised.

13. Anomalies of Venous Return: These anomalies may occur in either the pulmonary or the systemic circulation. In other words, some or all of the pulmonary veins may empty into the right auricle or into veins that drain into the

right auricle, and part or all of the blood returning through the venae cavae may empty into the left auricle.

If all the pulmonary veins drain into the right auricle or its tributaries, there must be an interauricular defect to make the condition compatible with life. In most patients in whom several but not all of the pulmonary veins drain into the right auricle or its tributaries there are associated malformations. In one of the patients studied by Dr. Bing[48] in whom several but not all of the pulmonary veins emptied into the superior vena cava but in whom there were no associated malformations, there was a great increase in pulmonary blood flow without an associated increase in the pulmonary pressure. The ideal in surgical therapy in such a case would be the anastomosis of the aberrant veins to the left auricular appendage or one of its tributaries. Almost equally good results would probably be accomplished by the less difficult procedure of making a large interauricular defect. Muller[49] has recently anastomosed the side of an anomalous left pulmonary vein to the left auricular appendage with a good result. Dr. Bing has found pronounced pulmonary arterial hypertension with strain on the right ventricle in two patients in whom all of the pulmonary veins drained into the right auricle through the superior vena cava. The systemic arterial oxygen saturation in these patients was only moderately depressed, and the main problem was the burden on the right ventricle. An attempt was made in one of these patients to correct the venous return but without success. The creation of a very large interauricular defect would probably have relieved the strain on the right ventricle. The larger the auricular defect the greater will be the opportunity for a right to left shunt.

As pointed out earlier, there is danger of spontaneous closure of an anastomosis between a vein and an auricle or auricular appendage. The anatomic structure is such that it is difficult if not impossible to anastomose the right pulmonary veins to the left side of the heart unless they actually pass across the midline and into the left chest. In some instances all of the pulmonary veins, right and left, do come together in the left side of the chest and enter the innominate vein or the superior vena cava by a common trunk. Under such circumstances it is important to realize that one is dealing with a common trunk and that total occlusion while one is performing an anastomosis to the auricular appendage would result fatally. If the trunk is large, it may be partially occluded while the anastomosis is being performed; otherwise one of its branches may be used for this purpose. As indicated previously, the implantation of the anomalous vein into the correct side of the heart is the ideal procedure, but the dangers of subsequent closure of the anastomosis and the difficulties of determining the exact anatomic picture may influence one to make a large interauricular defect if one is not already present.

Important anomalies of the venae cavae are less frequently observed. If an anomaly is discovered that is producing symptoms, the problems in treatment are similar to those that have been discussed in connection with the pulmonary veins; that is, transplantation into the proper side of the heart or the making of a large interauricular defect. Bilateral superior venae cavae occur

fairly frequently and are usually associated with other malformations. Usually both of the superior venae cavae enter the right side of the heart, but in some instances the left one drains into the left side. If this latter condition as well as the presence of a normal right superior vena cava is determined preoperatively, one may ligate the left superior vena cava in the course of attacking the associated congenital malformation.

III. DISORDERS IN THE TREATMENT OF WHICH SURGERY IS OF DOUBTFUL VALUE OR SUITABLE METHODS ARE NOT YET FULLY DEVELOPED

A. Acquired.—

14. Coronary Artery Disease: I hope that I am in error in placing this disorder in this group because if it is proved that many cases of coronary arterial disease are amenable to surgical therapy, it will constitute one of the greatest of all advances in medicine. However, it would appear that our chief hope lies in the prevention of arteriosclerosis, possibly by dietary measures.

It is largely due to the ingenuity and tenacity of Dr. Claude Beck[50, 51] that some surgical progress has been made in this most difficult field. In many experiments and some clinical trials, efforts have been made to protect the heart against coronary occlusion by the establishment of an extracoronary bed and of communications between the three main coronary arteries. The methods which have been employed include the grafting of adjacent tissues such as skeletal muscle, lung,[52] pericardium, and omentum onto the surface of the heart in an effort to supply blood to the ischemic myocardium from an outside source; the use of chemical or physical inflammatory agents applied directly to the heart in an effort to stimulate intercoronary communications; and more recently attempts to cause a backward flow of blood through the coronary veins and capillaries by a direct anastomosis between a systemic artery and the coronary sinus of the heart.[51, 53] Other efforts which have been made to improve the condition of the patient with coronary arterial disease and angina pectoris include thoracic sympathectomy for the relief of pain, ligation of the great cardiac veins, which may cause stasis and a better distribution of blood in the myocardium, total thyroidectomy, the implantation of a systemic artery into the myocardium, and the excision of infarcts of the heart.

The operation which appears to me to have the most likely possibilities for improving the circulation of the myocardium is the original one proposed by Beck[50] in which some tissue adjacent to the heart is placed against the heart and an inflammatory reaction is provoked by an agent such as talc. In addition to the fact that this is a less dangerous procedure than that in which an anastomosis between a systemic artery and the coronary sinus is created, it would seem that the chances for prolonged improvement in the circulation are greater. Whether one uses total, partial, or no occlusion of the coronary sinus at its ostium in conjunction with its anastomosis to a systemic artery, it is likely that the shunted arterial blood will ultimately find its way into the heart chambers through dilated venous channels in which the pressure is lower than that in the capillaries.[54, 55] I am skeptical of the value of the systemic artery

to the coronary sinus operation, despite the fact that Heimbecker and I[56] have shown recently by observation of capillaries and determinations of the oxygen content of blood that arterial blood can be made to flow backward through the veins and capillaries of the intestinal tract, for a brief period at least, under conditions which will not be detailed here. Fluids seek a low pressure route and blood will not flow through a high pressure zone for an extended period when small veins exist which are capable of dilation when subjected to increased pressure.

Beck has emphasized his belief that the operations which he has described will not eradicate the disease and that a complete cure is not to be expected. Since the average duration of life of patients who survive the first attack of coronary occlusion is approximately five years, it is extremely difficult to evaluate the effects of a procedure which is not curative, and thousands of trials will be required before a definite answer can be supplied. Even though it would appear that the possibilities of making real progress in the treatment of coronary disease by existing techniques are not great, the importance of the problem demands that experimentation be continued.

15. Aortic Valvular Stenosis: Aortic stenosis as an isolated lesion is rare and is more often acquired than congenital. Rheumatic fever is the most frequent causative agent. It is fortunate that the lesion is less common than mitral stenosis, for the surgical problem is thus far a more difficult one. Our efforts to treat aortic stenosis are limited to two cases and both resulted fatally from ventricular fibrillation. The first patient (who was to be operated upon by the late Horace Smithy) died as the incision was being made in the chest wall, and the second died shortly after the stenotic valve had been incised and dilated through an incision in the left ventricle. Bailey[31] has attempted to stretch the stenotic orifice by the use of a dilator introduced through the carotid artery and down the aorta, but he has now abandoned this method in favor of the ventricular approach.

Subaortic stenosis,[57] which is also rare, is usually a congenital lesion and is caused by the persistence of a band or membrane of connective tissue which lies immediately below the aortic valves. The problem of surgical therapy should be easier in this condition than in rheumatic aortic stenosis.

16. Insufficiency of Heart Valves: The problem of the surgical treatment of insufficiency of the various valves of the heart is still decidedly in the experimental stage and will probably remain there for some time. One could probably anchor in place an artificial mechanical valve by the use of existing techniques, but there is great doubt whether such a valve would function for an extended period. If one is to place accurately a graft of viable tissue such as pericardium, one should be able to suture with care in a dry field under direct vision, which means that an extracorporeal circulation is required. Furthermore, it remains to be determined whether such valves will live and function properly. This latter problem may present greater difficulties than those of artificial circulation. Gordon Murray and his associates[58] used vein segments, and Templeton and Gibbon[59] employed vein and pericardial grafts in

efforts to repair experimental defects in valves. The results are moderately encouraging, particularly with the use of pericardium, but the observation period is too short for critical assessment. Even if the results in partial replacement of the normal valve by a graft were practically perfect, the question is still unanswered as to the fate of a graft placed in a scarred area such as exists in an acquired insufficiency. The functioning of heart valves is an extremely complicated mechanism and is dependent upon the attachment of the chordae tendinae as well as other factors. At any rate, the problem of valvular insufficiency is an important one which it will be very difficult to solve.

I am aware of the fact that Bailey and his associates are placing a pedicled flap of pericardium across the left ventricle just below the mitral valve in the treatment of mitral insufficiency, and it is said that the early results are encouraging. A long observation period will be required for proper evaluation.

17. Intracardiac Tumors: Existing surgical methods are suitable for the excision of tumors of the surface of the heart. Unfortunately these lesions are usually metastatic. The proper treatment of tumors of the interior of the heart awaits the development of more accurate means of diagnosis as well as means either for good visualization with instrumentation of the interior of the beating heart, or perfection of the artificial circulation which will permit an open operation in a bloodless chamber of the heart.

B. Congenital.—

18. Anomalous Origin of Left Coronary Artery: In the rare anomaly in which the left coronary artery arises from the pulmonary artery the infants usually die of cardiac failure during the early months of life. This condition may occur as the sole anomaly. Bland, White, and Garland[60] have shown that the condition can sometimes be diagnosed by the finding by electrocardiography of inversion of the T waves in all three leads combined with low voltage curves.

There is a possibility but not a great likelihood that this condition may be treated surgically by severing the connection of the left coronary with the pulmonary artery and anastomosing its distal end to a systemic artery such as the subclavian. It would be necessary to perfuse the coronary artery with blood while the procedure is being performed. Smith and I have made a few attempts to carry out a similar procedure in experimental animals but thus far without success. The small size of the coronary artery in an infant renders perfusion and simultaneous anastomosis a difficult and dangerous undertaking.

19. Auricular Septal Defects: A defect of the auricular septum is a common lesion and frequently an isolated one. These defects vary greatly in size and position. The shunt of blood is usually from the left to the right auricle and hence there is usually no cyanosis. The shunt imposes an added load on the right side of the heart, and there is generally dilatation of the right atrium and ventricle and right ventricular hypertrophy. Failure of the right side of the heart may occur. In some cases the shunt is reversed, that is, from right to left, and cyanosis is present. In the absence of pulmonary hypertension

it seems obvious that a more balanced circulatory pattern would result from closure of an isolated auricular septal defect.

When pulmonary hypertension is present it may very well be that closure of an interauricular defect is contraindicated. The answer probably hinges on the question whether the pulmonary hypertension is primary or secondary, and if secondary, whether the hypertension would be lessened by closure of the defect. In the presence of marked arteriolar disease in the lungs the right ventricular pressure and consequently the right auricular pressure are elevated. As a result the shunt in the two auricles may be directed from right to left. Under these circumstances repair of the auricular defect would close the opening which may be preventing an excessive increase in pressure in the right auricle and may transform the condition into one resembling Ayerza's disease. Since the cause of pulmonary hypertension in association with an auricular septal defect is obscure and may be due to primary changes in the pulmonary arteries, I simply wish to emphasize the possibility that closure of the defect may be contraindicated. It is possible that closure of the defect combined with denervation of the lungs may be the treatment of choice. Denervation of the lungs without closure of the defect might make the condition worse.

Since there are many auricular septal defects unassociated with complicating factors, it is important that a satisfactory method for closing them be developed. Much experimental work along this line is being performed, such as that of Cohn[61] on the use of part of the wall of the auricle, and that of Swan[62] on the employment of the auricular appendages. One of the difficulties of the problem is that experimentally produced defects are prone to spontaneous closure, and it is difficult to gauge the effectiveness of various methods of repair. The method which Hanlon and I[36] devised for creating an interauricular defect usually results in one which does not close spontaneously, but its position is not the same as that of naturally occurring interauricular defects. Gordon Murray[63] made a preliminary report on his attempts to close interauricular defects by a blind method in which coarse suture material is passed through the heart in the region of the septum and is tied loosely. A somewhat similar method has been reported by Perianes,[64] I doubt whether any of the clinical or experimental methods which have been reported thus far will prove to be the answer to this problem. Good visualization of the interior of the auricles is desirable since the defects vary so greatly in size and position, and a wholly satisfactory method may have to await the perfection of the extracorporeal circulation.

20. The Lutembacher Syndrome: This syndrome consists of an auricular septal defect combined with congenital or acquired mitral stenosis and great dilatation of the pulmonary artery. It is well known that patients with auricular septal defects are prone to have rheumatic fever and mitral stenosis. The greatest strain in the Lutembacher syndrome is probably on the right auricle, the right ventricle, and the pulmonary artery, whereas the left ventricle and the aorta are spared except in the later stages. Because of the fact that the shunt is from left to right, there is usually no cyanosis. White[65] has pointed out that

some patients with this condition survive to old age, but this is the exception rather than the rule.

If surgical therapy is indicated, it would appear that the attack should be on the stenotic mitral valve rather than upon the auricular septal defect. If the flow of blood through the stenosed mitral valve is increased by commissurotomy, the left to right shunt would decrease, thus lessening the burden on the right side of the heart. If the mitral stenosis can be dealt with effectively, and if pulmonary arterial disease is not present, it would probably be desirable to close the interauricular defect if a safe and suitable method were available. In the presence of pulmonary arteriolar disease the defect should not be closed even if the mitral stenosis has been alleviated since the defect probably serves as a safety outlet in permitting some right to left shunt and prevents an excessive rise in pressure in the right auricle. As stated previously, the pulmonary artery in the Lutembacher syndrome is greatly enlarged. Even if one could reduce the size of this vessel by surrounding it with an irritating material such as cellophane, it is doubtful whether the patient would be benefited.

Approximately one year ago I performed a mitral commissurotomy on a patient ill with the Lutembacher syndrome. She survived the procedure, but her condition is little if any improved at the present time.

21. Ventricular Septal Defects: Defects of the ventricular septum are frequently observed lesions, they are difficult to diagnose with certainty, they vary greatly as to size, location, and associated symptoms if any, and many of them are compatible with long life. The shunt of blood is usually from left to right. Since this shunted blood returns to the left side of the heart, both ventricles take part in transporting it. In this respect the situation is different from that in interauricular defects with a left to right shunt in that the right ventricle has the entire burden of pumping the shunted blood.

The prognosis in patients with small ventricular septal defects is excellent, and surgical therapy is not indicated. It is in patients with large high defects in whom the large shunt causes changes in the pulmonary blood vessels and in those in whom for some reason a right to left shunt develops that closure of the defect might be considered. In patients in whom a large shunt is predominantly left to right, closure of the shunt would undoubtedly benefit the patient by protecting the pulmonary vessels and reducing the strain on the heart. On the other hand, if pulmonary hypertension and a right to left shunt are present, closure of the defect might result in an increase in the residual volume of the right ventricle, a rise in pressure in the right ventricle and pulmonary artery, and in right-sided heart failure.

Ventricular septal defects are hard to produce experimentally and are likely to close spontaneously, which makes difficult the problems of surgical methods of closure. As far as I know, no satisfactory method has been developed. Murray[63] made a preliminary report of his efforts to close ventricular septal defects in patients by a blind suture method. Because of the fact that the exact position of the defect varies widely from patient to patient and that some of the

defects are in close proximity to heart valves and to the orifices of major arteries, I do not believe that a blind method of suture is the answer to this surgical problem. The defects which produce symptoms are those that are closest to the heart valves and great arteries, and these present the chief difficulty in closure. As indicated previously, the low ventricular defect is a relatively benign lesion and closure is rarely if ever indicated. I am fairly certain that the proper surgical closure of ventricular septal defects that are producing symptoms will have to await the further development of methods of carrying on the circulation artificially which will allow under direct vision the careful placing and suturing of a graft of tissue such as pericardium in a position where there will be no interference with the action of heart valves or blockage of the orifices of important vessels. These technical difficulties added to those of accurate anatomic diagnosis indicate that it will be some time before this problem is solved.

22. The Eisenmenger Complex: In the Eisenmenger complex there is a high ventricular septal defect; the aorta overrides this defect and thus receives blood from both the right and the left ventricle. It differs from the tetralogy of Fallot mainly in that there is no pulmonic stenosis. The pulmonary artery is usually very large, and pressure within it is high. This condition, which fortunately is not common, may not be detected in childhood since cyanosis may not appear until the time of adolescence or later.

Serious changes in the pulmonary arterioles have been described in the Eisenmenger complex. Old and Russel[66] found the following: (1) medial hypertrophy; (2) an increase in elastic tissue with respect to both thickening of the pre-existing fibers and the presence of new fibers between the elements of the media; and (3) nonatheromatous intimal thickening with proliferation of fibrous tissue. These changes result in varying degrees of narrowing of the smaller pulmonary vessels. It has not been determined with certainly whether the pulmonary hypertension is primary or secondary, but it is very likely that it is secondary to the large high ventricular defect. If it were not for this increase in the pulmonary vascular resistance, most of the output of both ventricles would go to the lungs and the systemic flow would be inadequate. According to Bing,[67] the blood flow through the lungs in the Eisenmenger complex is essentially normal in quantity.

The ideal operation for the treatment of the Eisenmenger complex would be the closure of the high defect before pronounced and irreversible alterations in the pulmonary arterioles occur. As indicated in the discussion of the treatment of other high interventricular defects, this operation should be performed in a dry field under direct vision, and a graft of some tissue such as pericardium should be used. The proximity of the aorta to the defect renders an attempt at blind closure too dangerous. After definite pulmonary hypertension and visible cyanosis have developed, closure of the defect might result in failure of the right side of the heart. Since the pulmonary blood flow in this condition is said to be normal, and since there is severe constriction of the smaller

pulmonary vessels, it is doubtful whether an artificial reduction in the size of the pulmonary artery would accomplish any good, and it might make the condition worse. This point, however, has not been determined. A significant reduction in the size of the main pulmonary artery combined with sympathectomy might result in improvement. Unfortunately, an entirely satisfactory method for the partial closure of large diseased arteries has not been developed, and there is always the danger of subsequent hemorrhage at the site if a sizable reduction in caliber is caused. All in all, the problem of the surgical treatment of the Eisenmenger complex and of high ventricular septal defects in general is a most difficult one. Its solution probably will have to await further developments in intracardiac surgery.

23. Pulmonary Hypertension in Association with Congenital Heart Disease: Pulmonary hypertension in association with a number of the congenital cardiovascular defects has already been discussed briefly. The question arises whether the alterations in the pulmonary arterioles are primary[68] or whether they are secondary to the congenital defect in the heart or great blood vessels. The future role of surgical therapy in the treatment of pulmonary hypertension in association with congenital heart disease is bound up so closely with this unsettled question of etiology that no dogmatic statements seem warranted. Even if it be granted that the pulmonary hypertension in the majority of cases is due directly to the alteration in circulatory dynamics caused by the cardiovascular defect, there is still the problem whether these changes in the pulmonary vessels will be reversed if the defect is corrected. These alterations in the pulmonary vessels are usually anatomic rather than vasospastic. It has been stated previously that in many cases the increase in the resistance in the pulmonary vascular bed serves as a safety mechanism in preventing an unbalancing of the systemic and pulmonary vascular systems. This is probably the case in the Eisenmenger complex, transposition of the great vessels, isolated ventricular septal defects, aortic atresia with patent ductus, the single ventricle in which both great arteries or the aorta alone arises from the rudimentary outflow chamber, and in some instances of truncus arteriosus. Even if the nature of the pulmonary vascular resistance were such that it could be lowered by sympathectomy without correction of the defect, severe circulatory disturbances would likely ensue. I am of the opinion that the pulmonary vascular changes are usually secondary to the cardiovascular defect and that the treatment should consist of a direct attack on the defect when and if a method is available. Naturally the best results will be obtained in those patients in whom vascular alterations in the lungs are not far advanced. If the primary defect can be remedied, it is possible that an additional procedure such as pulmonary sympathectomy will be helpful in reducing the pulmonary resistance, but there is no convincing evidence that the pulmonary vessels are under autonomic control. Unfortunately, surgical means are as yet unavailable for dealing with many of these intracardiac defects, and therapy will have to await further advances.

Discussion

Many significant advances in cardiovascular surgery can be and will be made by the employment of existing methods and techniques, but many others will have to await the further development of methods for carrying on the circulation artificially in order that one may operate under direct vision in a bloodless field. Still others are dependent upon the solution of the problem of homotransplantation of tissues. This latter problem is more baffling than the former, and no real strides have been made. It is to be hoped that some agent will be found that will so alter the donor-recipient relationships that transplanted tissues will survive and function permanently. The work with cortisone and ACTH along these lines is not very encouraging.

Important advances have been and are being made in studies on methods for carrying on part or all of the circulation artificially. Many of the needs for and future applications of these methods have been mentioned in this paper in the discussion of individual lesions.

Kolff and others who have worked on the artificial kidney have demonstrated conclusively that it is possible to withdraw blood continuously, to pass it through an artificial circuit, and to return it to the body without harm to the patient. The blood is removed from an artery of the patient, who has received heparin, is passed through the artificial kidney, and is returned to one of the patient's veins. The problem is more complicated if one wishes to take over the cardiorespiratory functions because the use of the artificial lung presents greater complexities than does that of the artificial kidney. In order to take over the cardiorespiratory functions, it is necessary to heparinize the patient, to withdraw blood from a vein, to expose it to oxygen and allow carbon dioxide to escape, and then to pump the oxygenated blood into a peripheral artery. As a result of the work of Gibbon and many others, oxygenators that function with remarkable efficiency have been constructed.

PARTIAL CIRCULATION

The mechanical lung and pump as partial replacement for the functions of the heart and lungs should be helpful to patients having difficulty in oxygenating adequately the blood which is to enter the systemic arterial system. Among other conditions, the procedure should prove helpful in the treatment of patients with severe pulmonary edema which cannot be relieved by conventional methods of treatment.

The remaining remarks on partial circulation will deal only with replacement of the function of the right or the left side of the heart, the subject's own lungs being used for the oxygenation of the blood. If and when the artificial lung is perfected and proved to be safe, it is likely that the use of methods for replacement of the function of one of the two sides of the heart will be limited to experimental work, since the cannulation of the great blood vessels is in itself an extensive and a dangerous procedure. Furthermore, there are many intracardiac and extracardiac procedures in which it is desirable to have both sides of the heart bloodless.

Leeds, Gray, and Cook,[69] Sewell and Glenn,[70] and others have rendered the *right side* of the heart bloodless, except for coronary venous return, by diverting the blood from the superior and inferior venae cavae to a pump and then through a cannula to the pulmonary artery. As indicated previously, this required the insertion of at least two large cannulae. Since the coronary blood flow equals 5 per cent or more of the cardiac output, difficulties are presented in operations on the right side of the heart from the viewpoints of visibility and blood loss. If the outflow from the coronary sinus and thebesian vessels is to be collected and returned to the circulation, it should be debubbled. Sufficient experimental evidence has been accumulated to demonstrate that a unilateral pump can be opened and closed with recovery of the animal.

A unilateral pump to replace the function of the right side of the heart temporarily should theoretically allow one to operate on lesions of the tricuspid and pulmonary valves, including stenosis, atresia, and insufficiency, on Ebstein's disease, on the rare infundibular stenosis unaccompanied by a septal defect, and on some intracardiac tumors. The unilateral pump would not suffice for the closure of septal defects or for operations for other lesions associated with septal defects. The pump will probably find its greatest usefulness in permitting one to create various experimental lesions of structures of the right side of the heart.

Zollinger,[71] Kantrowitz,[72] and others have used a unilateral pump to substitute for the *left side* of the heart. The blood from the left auricle or the pulmonary veins is withdrawn through a cannula and it is then pumped through another cannula into a systemic artery such as the subclavian. It is more difficult to substitute for the left side of the heart than the right. One of the reasons for this is the likelihood of arterial air embolism associated with opening the left side of the heart. Furthermore, the coronary arterial flow is dependent, when the left heart pump is used, upon the competence of the aortic valve. Nevertheless, Kantrowitz has reported survival of cats following opening and closing of the bloodless left ventricle.

A unilateral pump to replace temporarily the function of the left side of the heart should theoretically allow one to operate on lesions of the mitral valve and on intracardiac tumors. The coronary arteries would probably have to be cannulated and perfused if one were operating for aortic stenosis or insufficiency. The unilateral pump would not suffice for the treatment of septal defects. The present results on the treatment of mitral stenosis are so encouraging that the employment of a pump in this connection seems unlikely. Because of the dangers accompanying the use of the left heart substitute, and because of its limited clinical usefulness as visualized at present, it would appear that this method will be employed mainly in the creation of experimental lesions.

TOTAL CIRCULATION

Wesolowski[73] and others have shown that *both sides* of the heart may be rendered relatively bloodless by using two pumps, one for the right and another for the left. The connections made would be similar to those described

under partial circulation; namely, one apparatus to withdraw blood from the venae cavae and propel it to the pulmonary arterial circulation, and another to withdraw blood from the left auricle or pulmonary veins and pump it to a systemic artery. Under these conditions the entire heart is deprived of blood except for the coronary flow, and various intracardiac procedures would be possible. It seems doubtful, however, whether the use of two pumps to replace the heart's functions will ever be a practical procedure in patients because the connections necessary between the two pumps and the patient make it too formidable. The method should continue to serve a useful purpose in experimental work.

Another method by which the function of the heart and lungs can be supplanted is by *cross circulation* between the recipient and a donor. This can be accomplished by having the donor's heart serving as the pumping mechanism, or mechanical pumps can be used. In either case, the donor's lungs would oxygenate the blood. Experiments indicate that the dangers to the donor are about as great as those to the recipient, and it would appear that this method will not have clinical application.

The last and best method is that in which an *artificial oxygenator* is used. The method involves cannulation of the venae cavae and passing of the venous blood through an oxygenator or artificial lung. The blood which has taken up oxygen and has lost carbon dioxide is then pumped to a systemic artery. Both sides of the heart are rendered relatively bloodless (if the aortic valve is competent) and intracardiac procedures such as operations on heart valves, septal defects, and intracardiac tumors are possible. The cannulation is simple as compared to that which is required when an oxygenator is not used. Those who are working in this field have had many problems to solve such as the prevention of damage to the blood and the prevention of air embolism. The two major problems remaining are the construction of a simple suitable oxygenator and the solving of the difficulties and dangers of operating on heparinized patients. Under the leadership of investigators such as Gibbon, Crafoord, Bjork, Kolff, Dennis, Glenn, Jongblood, and others, it is very likely that these problems will be solved. Only when this time arrives will it be possible to determine how well the diseased human heart will withstand having its functions taken over temporarily and incisions made into its chambers. It is probably fortunate that the dog's heart, because of its great tendency to fibrillate, presents a rigid test for the artificial circulation; the human heart presents less of a problem in this respect. It would seem that the initial clinical employment of the artificial heart and lungs for total replacement should be on patients with a usually fatal disorder such as congenital transposition of the aorta and pulmonary artery. The hearts of these patients withstand operative procedures remarkably well, and the use of the artificial circulation should give one an opportunity to divide and transpose the aorta and the pulmonary artery.

I hope it is obvious from my remarks that there are many unsolved problems in cardiovascular surgery. Many of these will be solved by modifications

in existing techniques. Others will have to await further fundamental developments. With the interest and enthusiasm that are being shown by competent investigators, the future development of this field of surgery seems assured.

REFERENCES

1. Report of National Conference on Cardiovascular Diseases. Published April, 1950 by the American Heart Association.
2. Delorme: Sur un traitement chirurgical de la symphyse cardo-pericardique, Bull. et mém. Soc. de chir. de Par. **24:** 918, 1898.
3. Holman, E.: The Recognition and Correction of Constrictive Pericarditis, J. THORACIC SURG. **18:** 643, 1949.
4. Blalock, A., and Ravitch, M. M.: A Consideration of the Nonoperative Treatment of Cardiac Tamponade Resulting from Wounds of the Heart, Surgery **14:** 157, 1943.
5. Elkin, D. C., and Campbell, R. E.: Cardiac Tamponade: Treatment by Aspiration. Presentation before Southern Surgical Association, December 5, 1950, Ann. Surg. In Press.
6. Clay, R. C., and Blalock, A.: Congenital Arteriovenous Fistulas in the Mandible, Surg., Gynec. & Obst. **90:** 543, 1950.
7. Blakemore, A. H.: Personal communication, 1951.
8. Gross, R. E.: Surgical Treatment for Coaretation of the Aorta. Experiences from 60 Cases, J. A. M. A. **139:** 285, 1949.
9. Lam, Conrad: Personal communication, 1951.
10. Cutler, E. C., and Beck, C. S.: Present Status of Surgical Procedures in Chronic Valvular Disease of the Heart; Final Report of all Surgical Cases, Arch. Surg. **18:** 403, 1929.
11. Allen, D. S., and Graham, E. A.: Intracardiac Surgery—A New Method, J. A. M. A. **79:** 1028, 1922.
12. Smithy, H. G., Boone, J. A., and Stallworth, J. M.: Surgical Treatment of Constrictive Valvular Disease of the Heart, Surg., Gynec. & Obst. **90:** 175, 1950.
13. Bailey, C. P., Glover, R. P., and O'Neill, T. J. E.: The Surgery of Mitral Stenosis, J. THORACIC SURG. **19:** 16, 1950.
14. Harken, D. E. Ellis, L. B., and Norman, L. R.: The Surgical Treatment of Mitral Stenosis. II. Progress in Developing a Controlled Valvuloplastic Technique, J. THORACIC SURG. **19:** 1, 1950.
15. Souttar, P. W.: Surgical Treatment of Mitral Stenosis, Brit. M. J. **2:** 603, 1925.
16. Sweet, R. H., and Bland, E. F.: Surgical Relief of Congestion in the Pulmonary Circulation in Cases of Severe Mitral Stenosis, Ann. Surg. **130:** 384, 1949.
17. Ellis, L. B., and Harken, D. E.: Mitral Stenosis, Clinico-Physiologic Correlations, with Particular Reference to Surgical Intervention. Trans. Am. Clin. & Climat. Assn., Vol. 60, 1948.
18. Munro, J. C.: Ligation of the Ductus Arteriosus, Ann. Surg. **46:** 335, 1907.
19. Graybiel, A., Strieder, J. W., and Boyer, N. H.: Attempt to Obliterate Patent Ductus Arteriosus in Patient with Subacute Bacterial Endarteritis, Am. Heart J. **15:** 621, 1938.
20. Gross, R. E., and Hubbard, J. P.: Surgical Ligation of Patent Ductus Arteriosus: Report of First Successful Case, J. A. M. A. **112:** 729, 1939.
21. Dammann, F.: Personal communication, 1951.
22. Keith, J. D., and Forsyth, C.: Aortography in Infants, Circulation **2:** 907, 1950.
23. Gross, R. E.: Complete Surgical Division of the Patent Ductus Arteriosus. A Report of Fourteen Successful Cases, Surg., Gynec. & Obst. **78:** 36, 1944.
24. Blalock, A.: Operative Closure of the Patent Ductus Arteriosus, Surg., Gynec. & Obst. **82:** 113, 1946.
25. Scott, H. W., Jr.: Closure of the Patent Ductus by Suture-Ligation Technique, Surg., Gynec. & Obst. **90:** 91, 1950.
26. Crafoord, C., and Nylin, G.: Congenital Coaretation of the Aorta and Its Surgical Treatment, J. THORACIC SURG. **14:** 347, 1945.
27. Blalock, A., and Park, E. A.: The Surgical Treatment of Experimental Coaretation (Atresia) of the Aorta, Ann. Surg. **119:** 445, 1944.
28. Blalock, A., and Taussig, H. B.: The Surgical Treatment of Malformations of the Heart in Which There Is Pulmonary Stenosis or Pulmonary Atresia, J. A. M. A. **128:** 189, 1945.
29. Brock, R. C.: The Surgery of Pulmonary Stenosis, Brit. M. J. **2:** 399, 1949.
30. Brock, R. C., and Campbell, M.: Infundibular Resection or Dilatation for Infundibular Stenosis, Brit. Heart J. **12:** 403, 1950.
31. Glover, R. P., Bailey, C. P., and O'Neill, T. J. E.: Surgery of Stenotic Valvular Disease of the Heart, J. A. M. A. **144:** 1049, 1950.

32. Barrett, N. R., and Daley, R.: A Method of Increasing the Lung Blood Supply in Cyanotic Congenital Heart Disease, Brit. M. J. **1**: 699, 1949.
33. Taussig, H.: Personal communication, 1951.
34. Blalock, A.: Surgical Procedures Employed and Anatomical Variations Encountered in the Treatment of Congenital Pulmonic Stenosis, Surg., Gynec. & Obst. **87**: 385, 1948.
35. Potts, W. J., Smith, Sidney, and Gibson, S.: Anastomosis of Aorta to Pulmonary Artery; Certain Types in Congenital Heart Disease, J. A. M. A. **132**: 627, 1946.
36. Blalock, A., and Hanlon, C. R.: Interatrial Septal Defect—Its Experimental Production Under Direct Vision Without Interruption of the Circulation, Surg., Gynec. & Obst. **87**: 183, 1948.
37. Taussig, H. B., Padmarati-Eyra, and King, J. T.: Personal communication, 1951.
38. Sellors, T. H.: Surgery of Pulmonary Stenosis—A Case in Which the Pulmonary Valve Was Successfully Divided, Lancet, p. 988, 1948.
39. Blalock, A., and Kieffer, R. F., Jr.: Valvulotomy for the Relief of Congenital Valvular Pulmonic Stenosis With Intact Ventricular Septum. Report of Nineteen Operations by the Brock Method, Ann. Surg. **132**: 496, 1950.
40. Bing, R. J.: Personal communication, 1951.
41. Varco, R.: Personal communication, 1951.
42. Neuhauser, E. B. D.: The Roentgen Diagnosis of Double Aortic Arch and Other Anomalies of the Great Vessels, Am. J. Roentgenol. **56**: 1, 1946.
43. Gross, R. E.: Surgical Relief for Tracheal Obstruction from a Vascular Ring, New England J. Med. **233**: 586, 1945.
44. Evelyn, K. A., Alexander, F., and Cooper, S. R.: Effect of Sympathectomy on Blood Pressure in Hypertension. A Review of Thirteen Years' Experience at the Massachusetts General Hospital, J. A. M. A. **140**: 592, 1949.
45. Blalock, A., and Hanlon, C. R.: The Surgical Treatment of Complete Transposition of the Aorta and the Pulmonary Artery, Surg., Gynec. & Obst. **90**: 1, 1950.
46. Taussig, H. B., and Bing, R. J.: Complete Transposition of the Aorta and a Levoposition of the Pulmonary Artery: Clinical Physiological and Pathological Findings, Am. Heart J. **37**: 551, 1949.
47. Brown, M., and Blalock, A.: Unreported Results on Experimental Transposition of Veins, 1951.
48. Friedlich, A., Bing, R. J., and Blount, S. G., Jr.: Physiological Studies in Congenital Heart Disease. IX. Circulatory Dynamics in the Anomalies of Venous Return to the Heart Including Pulmonary Arteriovenous Fistula, Bull. Johns Hopkins Hosp. **86**: 20, 1950.
49. Muller, W. H., Jr.: Personal communication, 1950.
50. Beck, C. S.: The Development of a New Blood Supply to the Heart by Operation, Ann. Surg. **102**: 801, 1935.
51. Beck, C. S., Staton, E., Batiuchok, W., and Leiter, E.: Revascularization of Heart by Graft of Systemic Artery into Coronary Sinus, J. A. M. A. **137**: 436, 1946.
52. Carter, B. N.: Surgery of the Heart and Allied Great Vessels, J. A. M. A. **138**: 1207, 1948.
53. Roberts, J. T., Browne, R. S., and Roberts, G.: Nourishment of the Myocardium by Way of the Coronary Veins, Fed. Proc. **2**: 90, 1943.
54. Stenstrom, J. D.: Vascularization of the Myocardial Capillary Bed by Arterialization of the Cardiac Veins, Canad. M. A. J. **59**: 420, 1948.
55. Johns, T. N. P., Sanford, M. C., and Blalock, A.: An Experimental Study of the Anastomosis of Arteries to the Coronary Sinus of the Heart of the Dog, Bull. Johns Hopkins Hosp. **87**: 1, 1950.
56. Heimbecker, R., Thomas, V.: and Blalock, A.: Experimental Reversal of Capillary Blood Flow, Circulation. In Press.
57. Taussig, H. B.: Congenital Malformations of the Heart, New York, 1947, Commonwealth Fund, p. 429.
58. Murray, G., Wilkinson, F. R., and MacKenzie, R.: Reconstruction of the Valves of the Heart, Canad. M. A. J. **38**: 317, 1938.
59. Templeton, J. Y., III, and Gibbon, J. H., Jr.: Experimental Reconstruction of Cardiac Valves by Venous and Pericardial Grafts, Ann. Surg. **129**: 161, 1949.
60. Bland, E. F., White, P. D., and Garland, J.: Congenital Anomalies of the Coronary Arteries; Report of an Unusual Case Associated with Cardiac Hypertrophy, Am. Heart J. **8**: 787, 1933.
61. Cohn, R.: An Experimental Method for the Closure of Interauricular Septal Defects in Dogs, Am. Heart J. **33**: 453, 1947.
62. Swan, H., Maresh, G., Johnson, M. E., and Warner, G.: The Experimental Creation and Closure of Auricular Septal Defects, J. THORACIC SURG. **20**: 542, 1950.
63. Murray, G.: Closure of Defects in Cardiac Septa, Trans. Am. Surg. Assn. **66**: 524, 1948.
64. Perianes, I.: Personal communication, 1950.

65. White, P. D.: Heart Disease, ed. 3, New York, 1944, The Macmillan Company, p. 295.
66. Old, J. W., and Russell, W. O.: Necrotizing Pulmonary Arteritis Occurring with Congenital Heart Disease, Am. J. Path. **26:** 789, 1950.
67. Bing, R. J., Vandam, L. D., and Gray, F. D., Jr.: Physiological Studies in Congenital Heart Disease. III. Results Obtained in Five Cases of Eisenmenger's Complex, Bull. Johns Hopkins Hosp. **80:** 323, 1947.
68. Edwards, J. E.: Functional Pathology of Certain Cardiovascular Malformations Which May Be Treated Surgically, Arch. Surg. **61:** 1103, 1950.
69. Leeds, S. E., Gray, N. N., and Cook, O. S.: Prolongation of Occlusion of the Cavae in Dogs by Diversion of the Circulation. I. Cannulation of the Superior Vena Cava, Surg., Gynec. & Obst. **91:** 399, 1950.
70. Sewell, W. H., Jr., and Glenn, W. W. L.: Experimental Cardiac Surgery. I. Observations on the Action of a Pump Designed to Shunt the Venous Blood Past the Right Heart Directly into the Pulmonary Artery, Surg. **28:** 474, 1950.
71. Sirak, H. D., Ellison, E. H., and Zollinger, R. M.: Cardiotomy into an Empty Left Ventricle, Surg. **28:** 225, 1950.
72. Kantrowitz, A., and Kantrowitz, A.: Experimental Artificial Left Heart to Permit Surgical Exposure of Mitral Valve in Cats, Proc. Soc. Exper. Biol. & Med. **74:** 193, 1950.
73. Wesolowski, S. A., Miller, H. H., Halkett, J. A. E., and Welch, C. S.: Experimental Replacement of the Heart by a Mechanical Extracorporeal Pump, Bull. New England Med. Center **12:** 41, 1950.

EXPERIMENTAL RECONSTRUCTION OF TRACHEAL AND BRONCHIAL DEFECTS WITH STAINLESS STEEL WIRE MESH

ROBERT M. BUCHER, M.D., W. EMORY BURNETT, M.D., AND GEORGE P. ROSEMOND, M.D.
PHILADELPHIA, PA.

THE purpose of this report is to present the results of some experimental work done on animals involving the reconstruction of tracheal and bronchial defects with wire mesh.

With recent advances in thoracic surgery, more diseases of the lungs are becoming amenable to surgical therapy, and the limits of operability have been greatly extended. In the case of malignant disease, however, there are still several barriers to the width of excision. One of these barriers is the trachea. If malignancy involves the tracheal wall, it is either inoperable or, at best, resection can be only palliative. If some method of reconstruction or anastomosis of the trachea were possible, then a segment could be resected just as a segment of the intestinal tract may be resected. Such a method would undoubtedly extend the curability of pneumonectomy for bronchiogenic carcinoma and would aid in the treatment of primary neoplasia of the trachea. There are many other possible applications as well.

Experimental work in this direction has been carried out in the past, and is doubtless being undertaken by many at the present time. Definite progress has been made, and it does not seem at all unlikely that eventually a method or methods of reconstruction of the trachea and bronchi will be added to the armamentarium of the thoracic surgeon.

REVIEW OF PREVIOUSLY REPORTED EXPERIMENTS

Probably the first report on experimental repair of the trachea was made by Hohmeier,[1] of Munich, in 1911. He excised windows of cervical trachea in dogs, and repaired these defects with fascia lata. In one animal a defect of 1.5 by 1.5 centimeters was made, and in another 2.5 by 2 centimeters. Neither of these developed any emphysema, and at the end of eight weeks there was a completely regenerated smooth mucosa with slight stenosis.

In 1912, Levit[2] reported a tracheal repair on a human patient. He closed a small tracheal fistula, which had resulted in an attempt at suicide, by freshening the edges and suturing fascia over the defect. The patient was well eight months after operation, although there was slight narrowing of the trachea.

In 1917, Neuhof[3] reported the closure of an old tracheotomy which had failed to heal by suturing fascia into the defect. This patient was well six

From the Department of Surgery, Temple University School of Medicine and Hospital.
Received for publication March 1, 1951.

months after operation and had no apparent stenosis, although direct laryngoscopy was not done. He also mentioned several other methods of reconstruction which had been used. One was to turn down a flap consisting of superficial layers of the thyroid cartilage and adjacent skin, with the skin inside. Another was the use of an osteocutaneous flap from the sternum or clavicle. Nothing was stated as to the results of these procedures. He also mentioned that circular resection of the trachea had been done with success, but stated that it was difficult to get accurate approximation. In addition, he mentioned a case of tracheal fistula which was cured by implantation of a sheet of silver filigree. Unfortunately, no further details were given.

Taffel,[4] in 1940, reported a group of experiments on dogs in which he used free fascial grafts to close various defects. In the first group, 1 by 1 centimeter defects were made in the cervical trachea and repaired with free grafts of deep cervical fascia. All of these were airtight, and none of the animals had any respiratory complications or subcutaneous emphysema. In the second group, 5 by 5 millimeter defects were made in the left upper lobe bronchus, and were repaired with a free graft of fascia from the chest wall. Again there were no respiratory difficulties. In the third group a left upper lobectomy was done, and fascia was sutured over the open bronchus. And in a fourth group the tracheal defect resulting from pneumonectomy was repaired with a free fascial graft.

These procedures were done in twenty dogs, all of which healed primarily. There was no encroachment on the caliber except in the case of the lobectomy closure. The graft did not seem to remain viable, but rather acted as a scaffold for the regeneration of fibrous tissue. Regeneration of the mucosa was evident at the edges of the defect in one week and complete by the end of the second week, and there were mucous glands present at the end of the third week. In none of these was there evidence of regeneration of a muscularis mucosa, and, although there were islands of cartilage reformed, these did not take part in filling the defect. By the end of the fourth week, practically all healing had taken place. Microscopy revealed an inner layer of pseudostratified ciliated epithelium, a submucosa with occasional mucous glands and a few islands of cartilage but no muscularis mucosa, and an outer layer of supporting adult connective tissue.

It is true that these defects were quite small, but this report gave evidence that adequate functional regeneration of trachea and bronchi could take place.

Probably the most extensive study was reported by Daniel[5] in 1948. He first repeated Taffel's work, and in thirteen dogs made small 8 millimeter square windows. He then used three methods of repair. In several a fascial graft was used, in others the defect was closed with fibrin film, and in others he merely sutured muscle over the defect. In the first two groups he demonstrated regeneration of epithelium, although there was slight narrowing in the fascia group, and rather marked narrowing in the fibrin film group. In the third group, in which muscle was sutured over the defect, tissue protruded into the lumen, and there was some tracheal obstruction.

He then excised segments of thoracic trachea and tied the cut ends over glass, stainless steel, or vitallium tubes. This procedure was used in twenty-one dogs, of which six succumbed within five days. In the remaining fifteen he found that the tube was held firmly in place by fibrous tissue and granulation tissue, with one exception in which the tube had slipped down to the carina. In all cases the silk ligature, which had been used to tie the cut end around the tube, had cut through and was lying inside the lumen of the trachea against the tube. Islands of cartilage began to appear in three weeks and then grew slowly, and epithelium began to grow from the cut ends and was nearly complete in six months.

Daniel, in a third group, using eight animals, resected the right lung and the carina. He then tied the cut ends of the trachea and the left bronchus over a curved tube. All but two of these animals died of leakage, obstruction, or hemorrhage. The remaining two were sacrificed at six and fourteen months, respectively. In one, there was wheezing which gradually became worse. At six months there was almost complete regeneration of epithelium and, grossly, evidence of cartilage, but there was moderate constriction. In the other, the glass tube loosened and was removed bronchoscopically at eight months. There was slight narrowing noted at that time. At the end of fourteen months this dog had some wheezing, and at autopsy was found to have narrowing to one-half the diameter of the normal trachea. Epithelization was complete, and cartilage rings were present.

Hanlon[6] has recently reported on the use of gelatin sponge to repair several types of bronchial defects in dogs. He first demonstrated that, in vitro, gelatin sponge formed a relatively airtight barrier. In ten animals he made an incision in the anterior bronchial wall and wrapped gelatin sponge around the bronchus. This was successful in all cases. In the second group of ten dogs, small segments of the anterior bronchial wall were excised, up to 1 centimeter in width, and one-half the circumference. Again gelatin sponge was wrapped around the bronchus, using no sutures, but depending on its adhesive qualities alone. All but one of these animals survived. The one fatality was due to an infected pneumothorax on the third day. In a third group of fifteen dogs a lobectomy was done and the open end of the bronchus was closed by a cap of gelatin sponge, again without any sutures. Two animals died of empyema, but the remaining thirteen survived. After sacrificing the animals it was demonstrated that the repair would tolerate intrabronchial pressures much higher than those occurring normally. Microscopy demonstrated fairly rapid regeneration of epithelium, complete in several weeks. The gelatin sponge seemed to act as a scaffold for fibrous tissue regeneration, just as the fascial grafts had in previous experiments.

Since the completion of this present experimental work a very interesting group of cases has been published by Gebauer.[8] In cases of bronchostenosis he has used a graft of dermis laced with stainless steel wire to reconstruct the posterior bronchial wall. His technique consists, briefly, in incising the posterior bronchial wall at the stenotic area, allowing this to retract, then fashioning an oval-shaped graft of dermis which is laced with No. 28 wire to give it

some rigidity. This graft is sutured into place with wire sutures to form a new, contoured, posterior bronchial wall. This method has been used in four clinical cases, all of which have been followed for a year or more. In all cases there seems to be satisfactory or fairly satisfactory function of the reconstructed segment.

PRESENT EXPERIMENTS

The reports of previous experimental work have demonstrated several things clearly. One is that regeneration of the trachea will occur. It seems that the regenerated wall is not complete in all elements, but does have a good mucosa capable of normal function. There is also a tendency to regeneration of cartilage, although it is not yet clear whether or not this regenerated cartilage will function well in maintaining a semi-rigid airway. Another fact demonstrated is one that might well be anticipated, and that is that there is a great tendency to some stenosis since the new wall is predominantly fibrous tissue, which tends to contract and thus diminish the lumen.

With these things in mind, the present experimental study was undertaken, using stainless steel wire mesh. This material was selected for several reasons. It has been demonstrated that stainless steel wire is quite inert and produces very little irritation. As a suture material it has been left in wounds for many years with no untoward effects. In the form of mesh, it has been used to repair large fascia-deficient hernias of the abdominal wall. Jonas[7] has demonstrated experimentally that the mesh becomes infiltrated and incorporated into the surrounding tissue with little or no inflammatory reaction. Fibroblasts seem to grow into the interstices and thus fix the mesh as an integral part of the new section of the abdominal wall. It is not completely rigid, but seems to have sufficient stability to prevent any recurrence of herniation. If this same type of healing were to take place in the trachea it would then be an ideal substance, since it would mimic the function of the tracheal cartilages and maintain a semi-rigid airway and prevent stenosis. Also, wire mesh may be moulded into a tube of any size, and thus the necessity of having a supply of various lengths and diameters of tubes would be eliminated. It would have an advantage over the rigid metallic or glass tube since the wire mesh could be used outside the tracheal wall and would not take up valuable intraluminal space, nor necessitate future removal with the possibility of subsequent stenosis.

FIRST GROUP

In this first group, mongrel dogs were anesthetized with intravenous nembutal and the cervical trachea exposed through a longitudinal incision. Two types of defects were then made. In some, one-half the circumference was resected, and in others the entire circumference (a segment) was resected. All the defects were made to extend over at least five tracheal cartilages. These defects were considerably larger than any of those previously reported, with the exception of Daniel's group of intrathoracic cases. Wire mesh (No. 40 or No. 80) was then wrapped around the entire trachea to form a tube outside the

tracheal wall. No attempt was made to approximate the cut ends in the cases of segmental resections. Interrupted wire sutures were then used to suture the wire to itself, forming the tube, and to the trachea. The wire overlapped the edges of the defect by approximately two rings at each end, and in some cases the cut ends were sutured as well, in an attempt to prevent any retraction. The cervical fascia and muscles were then sutured over this repair and the skin closed.

No specific postoperative care was given, with the exception that a few were given systemic penicillin during a time when respiratory infections seemed to be quite prevalent among all the animals.

Some subcutaneous emphysema was anticipated since there had been no attempt at airtight closure, but surprisingly there was none.

SUMMARY OF RESULTS IN FIRST GROUP

There were ten animals included in this group with four deaths. One death was due to overdose of anesthesia, one from a massive wound infection on the sixth postoperative day, and two from pneumonia on the second and tenth postoperative days, respectively. It did not seem that any of these deaths were directly attributable to the type of operative procedure with the possible exception of the case of wound infection.

The remaining six dogs were sacrificed at periods varying from six weeks to five months. The one sacrificed at six weeks showed almost complete epithelization grossly. The two sacrificed at eight weeks showed grossly complete epithelization, and in one there was evidence of regeneration of cartilage. In one of these, one-half the circumference was resected and this trachea showed no loss of diameter and only slight evidence of scarring. In the other, a complete segment had been resected, and here there was very slight narrowing with a small area of granulation tissue on the anterior wall. However, there was an adequate lumen, and no evidence of respiratory obstruction. The dog sacrificed at four months, illustrated in Fig. 1, had an entire segment of trachea excised, and here there was complete epithelization except for some piling up of scar tissue anteriorly, which did not appreciably influence the size of the lumen. There was also regenerated cartilage (Fig. 2). The same results were found in the animals sacrificed at five months. Both of these had complete segments excised, and in both there was complete epithelization except for the piling up of scar tissue anteriorly. One of these is illustrated in Fig. 1.

In summary, epithelization appeared to be nearly complete in six weeks and complete in eight weeks. However, there was a tendency to an excess of scar tissue in the anterior portion of the wall in those dogs having entire segments resected. In only one of these was there any evidence of obstruction, and this was only wheezing. In two dogs the wire had been crushed with consequent diminution of the lumen. It is believed that this occurred from being picked up by their chain collars. This complication, although not too likely to occur, must definitely be kept in mind. Probably some remoulding could have been

done endoscopically or by reoperation. In all the cases the wire mesh had been infiltrated by fibrous tissue, and appeared to maintain the newly formed wall as a semi-rigid tube.

It was difficult to obtain sections for microscopic study since the wire could not be sectioned and tissue could be obtained only by stripping it away from the

Fig. 1.—Left. Entire segment six rings (2.1 cm.) resected. Sacrificed at five months. Right. Entire segment five rings (2.3 cm.) resected. Sacrificed at four months. In both there is almost complete regeneration with re-epithelization except for slight piling up of granulation tissue anteriorly.

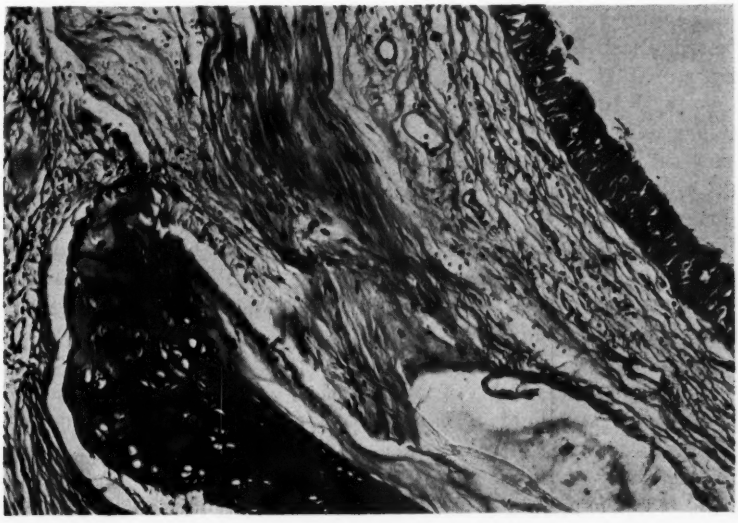


Fig. 2.—Photomicrograph of regenerated wall (200×) after four months.

mesh, to which it was quite adherent. Even so there was microscopic proof of normal respiratory epithelium in several. There was also both gross and microscopic evidence of regenerated cartilage.

SECOND GROUP

In the second group, mongrel dogs were anesthetized with intravenous nembutal, an endotracheal catheter was inserted, and artificial respiration given with intermittent positive pressure air. The right chest was then opened and the thoracic trachea exposed and freed, usually after sectioning the azygos vein. Similar segments of thoracic trachea were then excised, consisting of either one-half the circumference or the entire segment. Wire mesh was then wrapped around this defect with no attempt being made to approximate the cut ends. Again this mesh was sutured to the trachea and to itself with interrupted wire sutures. In the first case the parietal pleura was sutured over the reconstruction. This obviously did not form an airtight closure, and therefore several other methods of obtaining airtightness were tried. Free grafts of pericardium or skin were placed around the wire mesh and sutured to form an airtight covering. The chest was then closed and the pleural space aspirated. In most of these cases penicillin was given both by local instillation and systemically for a period of several days.

SUMMARY OF RESULTS IN SECOND GROUP

In this second group of eight animals, in which segments of thoracic trachea were excised and repaired, the results were rather disappointing since only one animal survived more than nine days. In the first animal, the repair was covered with pleura and this allowed leakage almost immediately with rapid demise of the animal. In the second animal, a graft of pericardium was used and this resulted in a good repair. However, in subsequent animals this could not be reduplicated. One animal died on the second postoperative day of pneumonia, with what was still an airtight repair. Another, was sacrificed on the sixth postoperative day since the wire mesh had not overlapped the cut ends of trachea sufficiently and had slipped, so that one edge of the mesh jutted into the tracheal lumen and caused obstruction. In this repair, however, there seemed to be no leakage. The remaining four deaths were due to leakage. In one, immediate leakage produced anoxia which led to cardiac arrest; in another, death occurred on the third day; in another, on the seventh day; and in the fourth, on the ninth day. The dog has a completely unstable mediastinum so that pneumothorax on one side results in a bilateral atelectasis and thus it seems that even the most minute leak will result fatally.

In one dog (see Fig. 3), a defect of four rings (1.5 centimeters), one-half the circumference was made. This was repaired with wire mesh covered by a free graft of pericardium. This dog recovered and remained completely well. After four and one-half months he was used for another experimental procedure, and at this time was intubated without difficulty. Following this he was sacrificed and it was found that there was absolutely no diminution of lumen. There appeared to be grossly complete epithelization and some regeneration of

cartilage. This illustration seems to show some bare wire mesh, but this is only because of transillumination. The mesh was infiltrated by fibrous tissue and, although the wall was not so thick as the regenerated cervical tracheal wall, it was completely adequate. Microscopy did not show epithelium, but this is not surprising since the epithelium was stripped away from the mesh, to which it was quite adherent, and thus was damaged making adequate sectioning impossible. This result was considered satisfactory for all practical purposes, since it was completely airtight, the mucosa smooth, and the lumen normal in caliber.

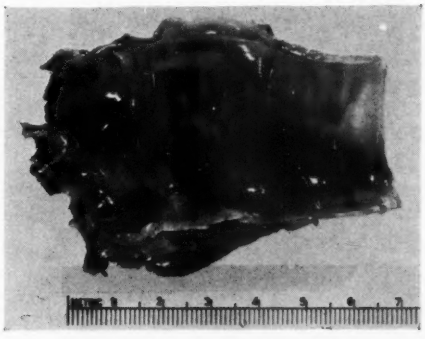


Fig. 3.—Defect of four rings, one-half the circumference of the thoracic trachea just superior to the carina. Sacrificed at four and one-half months. No narrowing and complete epithelization with beginning regeneration of cartilage. (Wire mesh is visible through intact epithelium.)

THIRD GROUP

In the third group, mongrel dogs were anesthetized with intravenous nembutal, an endotracheal catheter was inserted and artificial respiration given with intermittent positive pressure air. The right chest was then opened and a right pneumonectomy done. Either part or all of the carina was then resected so that a defect was formed which involved either the lateral half of the distal trachea and carina, or the entire distal trachea and carina. This was done to simulate a situation which is seen clinically. In some cases of carcinoma of the right bronchus there is extension to the carina or the lateral wall of the trachea. Such a lesion would be resectable if a method of anastomosing the trachea and left bronchus were available. These defects were then closed in a manner similar to that used in the second group, by making no attempt to approximate, but by bridging the defect with a tube of wire mesh. In this case the tube was tapered from the larger diameter of the trachea above to the smaller diameter of the left bronchus below. The wire mesh was found to be particularly suitable for this tapering. The mesh was sutured to the trachea and bronchus with interrupted wire, and then this area was wrapped with a full thickness skin graft which was sutured, or, in some cases, held by purse-string sutures at the ends and circular ligatures in the middle. Again in these cases the animals were given penicillin by local instillation and systemically for several days.

SUMMARY OF RESULTS IN THIRD GROUP

Again in this group the results were rather disappointing, since only one of the eight animals survived. One died of operative shock due to blood loss which occurred when the ligature on the pulmonary artery slipped. Two died of cardiac arrest, secondary to anoxia associated with leakage and obstruction, respectively. The remaining four deaths were due to leakage which occurred on the third or fourth postoperative day. Again, even a small leak would result in a pneumothorax with contralateral atelectasis.

Fig. 4.

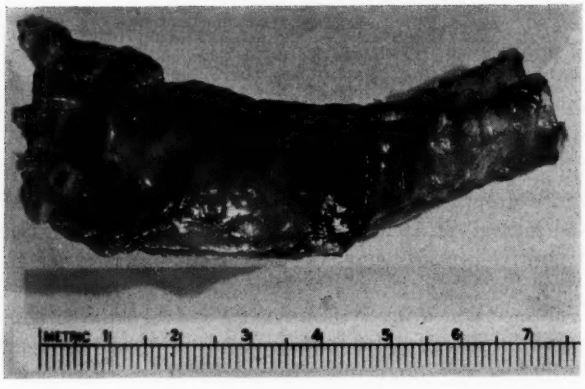


Fig. 5.

Figs. 4 and 5.—Right pneumonectomy with resection of one-half the carina and the right lateral half of the distal trachea to four rings proximal to the carina. Sacrificed at six months. Mucosal surface shows only beginning epithelization at edges with some bare wire mesh. External surface shows skin graft which apparently has been replaced by scar tissue. Repair completely airtight with no narrowing.

The survivor (see Figs. 4, 5, and 6) had a defect produced which included the lateral half of the circumference of the distal trachea and carina, extending to four rings above the carina. The wire mesh was placed and then covered with a full thickness skin graft which was sutured with interrupted wire. This animal remained completely well with no evidence of respiratory obstruction, and was sacrificed at six months. There was no diminution of the lumen, and the area of repair was quite clean. Regeneration had begun at the edges of the

defect but was not complete, and a small window of bare wire mesh remained, which was airtight because of its having been infiltrated by fibrous tissue. The skin graft appeared to have become a thin sheet of scar tissue, but microscopy demonstrated some of the dermal appendages still intact, although there was no demonstrable epithelium in the section studied. Although healing was not complete, this was still considered to be an entirely satisfactory result since the reconstruction functioned well and there was no diminution of lumen.

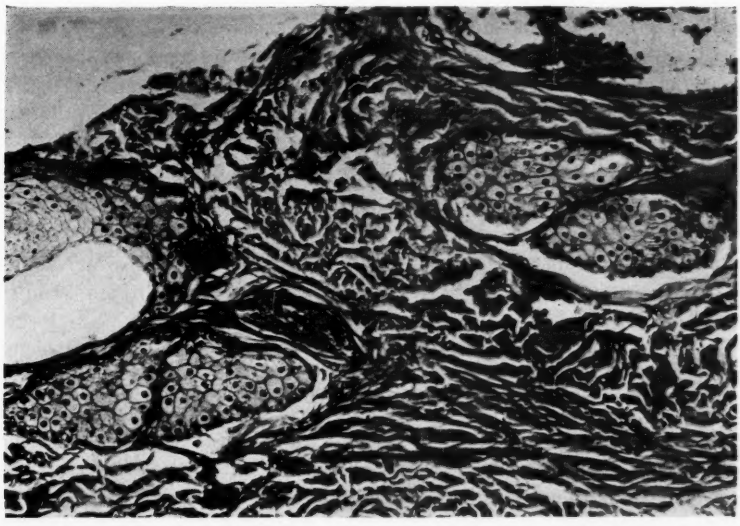


Fig. 6.—Photomicrograph of buried skin graft after six months. There is a fibrous stroma with scattering of chronic inflammatory cells, dilated structures resembling sweat glands, and a few sebaceous glands. No actual epithelium remains.

CLINICAL CASES

This method of repair has been used to a limited extent in two clinical cases which will be presented briefly.

CASE 1.—A 42-year-old woman was admitted to the hospital with an undifferentiated carcinoma of the thyroid which invaded the anterior wall of the trachea. A thyroidectomy was done, and a portion of the anterior wall of the trachea was resected. This defect in the trachea was repaired with no. 40 wire mesh by merely suturing it over the defect, overlapping the cut edges by approximately 1 to 1.5 centimeters. The wire was not wrapped completely around the trachea as had been done in the experimental animals. The wound was closed by suturing the pretracheal muscles and skin. A tracheotomy was done below this point of reconstruction. The patient had an uneventful convalescence and had no evidence of tracheal obstruction, wearing a plug in the tracheotomy tube. In four weeks this area was examined by direct laryngoscopy, and there appeared to be complete regeneration of normal epithelium. There was only slight diminution in calibre due to slight flattening anteriorly. This patient subsequently was found to have a pulmonary metastasis, and after six months there was local recurrence in the neck, with eventual reinvolvement of the trachea and obstruction, necessitating the use of the tracheotomy. Unfortunately, no autopsy was obtained after the patient's death.

CASE 2.—A 38-year-old man was admitted to the hospital with a tracheal fistula. He had had a tracheotomy which had been closed on two occasions, only to break down and reopen. The trachea was exposed and no. 40 wire mesh sutured over the defect. The pretracheal muscles and fascia were then sutured over this. The patient had an uneventful recovery and remained well for eight months, although he failed to return for follow-up after that time.

CONCLUSIONS

This method of repair of tracheal and bronchial defects which has been described has been used on a number of experimental animals and on two clinical cases. The results in the cervical trachea are rather encouraging. There were no deaths directly attributable to the type of procedure used, and regeneration of the tracheal wall and mucosa seemed to be at least functionally satisfactory. The wire mesh became an integral part of the regenerated segment, and maintained it in good functional position as a semi-rigid tube. In some cases there was an overgrowth of fibrous tissue along the anterior wall which occasionally produced a minor degree of obstruction. This seemed to occur only in those cases in which an entire segment was resected, and it is quite possible that if this were used clinically such redundancy could be corrected by endoscopic cauterization. The crushing which occurred in two animals due to rough handling is something not likely to occur in human beings, and could still possibly be corrected endoscopically or by reoperation. In the two clinical cases, the method seemed to work well.

There are several possible clinical applications. In cases of malignancy involving the tracheal wall, as illustrated in one clinical case, the wall could be resected and reconstructed. Also, it may be of benefit in the closure of tracheal fistulas as demonstrated by the second clinical case. It may also find application in cases of tracheal stenosis, where the entire segment could be excised and then reconstructed.

The results of its use in the thoracic trachea seem rather disappointing, since there were only two survivors in this experimental group. However, nearly all of these were due to leakage and, since the dog has such an unstable mediastinum, even the most minute leak is fatal. It is possible that this would be handled in the human patient by continuous aspiration until sealing, just as is done following some segmental lobectomies or for bronchopleural fistula following lobectomy. In the two animals that survived, the result was entirely satisfactory.

Although this method of repair could certainly not be advocated for trial in the thoracic trachea of the human patient at this early date in its experimental development, still one might postulate some possible applications. The most obvious application would be its use in malignancy to enable one to do a wider resection and then reconstruct any trachea which had been sacrificed. It is conceivable that this might be applied to bronchial lesions, such as bronchial stenosis, either by resecting an entire segment or part of the circumference of the stenotic area and reconstructing an adequate conducting passage. Also there have been a few cases in which bronchi have been eroded by compression

due to adjacent large lymph nodes. This wire reconstruction might be used merely to reinforce such an area of softening without actual resection.

In comparing this metthod to that of an intraluminal tube, the obvious advantage is that the wire mesh more closely approximates the function of the tracheal cartilages and is a part of the regenerated wall rather than a foreign body within the tracheal lumen. The disadvantages are just as obvious in that the wire mesh repair is undoubtedly more difficult and time-consuming than the ligation of cut ends over a solid tube. Also the difficulty in maintaining airtightness must be considered.

It may be that a combination of the two methods as suggested by Brown[9] would be more effective in the thoracic trachea. An intraluminal tube could be used to carry the patient over the time necessary for obtaining complete airtightness by fibrosis and then could be removed, leaving the wire mesh imbedded in the new wall and preventing later cicatricial contracture.

This paper is presented as a preliminary rather than a complete report. Additional work is being contemplated, and it is hoped that this present work will interest others to combine it with some of their studies, so that eventually there may be a relatively simple and satisfactory method of tracheal reconstruction to use in the human patient.

REFERENCES

1. Hohmeier, F.: A New Procedure for Closing Tracheal Defects, Munchen. Med. Wchnschr. **58**: 984, 1911.
2. Levit, H.: Repair of Tracheal Defects with Free Grafts of Fascia Lata, Arch. f. Klin. Chir. **97**: 686, 1912.
3. Neuhof, Harold: Fascia Transplantation into Visceral Defects, Surg., Gynec. & Obst. **24**: 383-427, 1917.
4. Taffel, Max: The Repair of Tracheal and Bronchial Defects with Free Fascia Grafts, Surgery **8**: 56-71, 1940.
5. Daniel, Rollin A. Jr.: The Regeneration of Defects of the Trachea and Bronchi. J. THORACIC SURG. **17**: 335-349, 1948.
6. Halon, C. Rollins: Observations on the Use of Gelatin Sponge in Closure of Experimentally Produced Defects of the Bronchus, Surg., Gynec. & Obst. **86**: 551-558, 1948.
7. Jonas, Karl C.: Stainless Steel Cloth as an Internal Prosthetic, Arch. Surg. in press.
8. Gebauer, Paul W.: Plastic Reconstruction of Tuberculous Bronchostenosis with Dermal Grafts, J. THORACIC SURG. **19**: 604-628, 1950.
9. Brown, Robert B.: Personal communication.

SHOULDER GIRDLE DYSFUNCTION FOLLOWING THORACOPLASTY COMBINED WITH PARTIAL SCAPULECTOMY*

A Study of Twenty-Three Patients During a Two-Year Period

Harry B. Doppelt, B.S., R.P.T., and Jacob Goldberg, M.D.
Castle Point, N. Y.

IN A recently published article,[1] the authors made some observations on the skeletal and muscular function changes resulting from thoracoplasty operations. These changes were classified under two headings: (1) scoliosis with concomitant changes in skeletal relationships of the head and neck, the shoulder girdle on the operated side, the thorax, and the pelvis; (2) skeletal deviations and changes of muscle function of the shoulder girdle on the operated side.

In this article the second of the two headings will be elaborated upon, as observed on those patients who have had both a thoracoplasty and a partial scapulectomy.

The findings contained herein should prove of interest and of value to thoracic surgeons, especially those who undertake this type of surgery, inasmuch as the resulting shoulder girdle dysfunction has been little studied heretofore.

PURPOSE, SCOPE, AND METHOD OF STUDY

Specifically, the purpose of the present study was to observe and record the disturbance of what has been called "the scapulohumeral rhythm"[2] and its resulting shoulder girdle disability on the operated side, as demonstrated in the movement of abduction of the humerus; also to note any changes in skeletal relationships of the shoulder girdle on the operated side. Toward these ends a Bucky roentgenogram was made of the scapulae in the anteroposterior view with the patient in a normal standing position, the arms hanging easily at the sides. In addition, goniometric measurements were taken of each patient's shoulder girdles.

A number of patients who had undergone a thoracoplasty were similarly studied to afford a control as to the changes which occur in the shoulder girdle after this type of surgery alone.

Measurements were made on the roentgenograms of the scapulae to determine the angle between the long axis of the humerus and the axillary border of the scapula, this angle being called the scapulohumeral angle. The goniometric measurements were utilized to record the range of abduction of both arms, the shoulder girdle on the nonoperated side serving as a control so that the degree of abduction loss on the operated side could be measured.

From the Physical Medicine Rehabilitation Service, Veterans Administration Hospital.
Received for publication Feb. 23, 1951.

*Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

GENERAL CONSIDERATIONS

The present study was made in a 605-bed Veterans Administration hospital for tuberculous patients during the two years between June, 1948, and July, 1950.

In this article patients who had both a thoracoplasty and a partial scapulectomy will be referred to as "scapulectomy patients." Those who underwent only a thoracoplasty will be referred to as "thoracoplasty patients."

Of the twenty-three scapulectomy patients studied, nineteen had all their surgery performed at this hospital. Two patients were operated upon elsewhere and came to this institution for convalescent care. The two remaining patients had only partial scapulectomies performed here.

The ten thoracoplasty patients included in the study are representative patients with uncomplicated postoperative courses, who were operated on at Castle Point (Table I).

TABLE I. TYPES OF SURGERY AND NUMBER OF EACH

THORACOPLASTY PATIENTS (10)	
One-Stage Thoracoplasty	
Following lobectomy, resection of portions of ribs, 2, 3, 4, 6	1
Two-Stage Thoracoplasty	
5 ribs resected	2
6 ribs resected	3
Three-Stage Thoracoplasty	
7 ribs resected	2
8 ribs resected	1
Four-Stage Thoracoplasty	
8 ribs resected	1
Total	10

SCAPULECTOMY PATIENTS (23)	PARTIAL SCAPULECTOMY DONE WITH LAST STAGE	PARTIAL SCAPULECTOMY DONE SEPARATELY	TOTALS
One-Stage Thoracoplasty (Resection of portions of ribs 2, 3, 4, 5)			
Following plombage	1		1
Following lobectomy		1	1
Conversion of extrapleural pneumothorax		1	1
Two-Stage Thoracoplasty			
5 ribs resected	1	2	3
6 ribs resected	7		7
7 ribs resected	1	1	2
Three-Stage Thoracoplasty			
6 ribs resected	1	1	2
7 ribs resected	3	2	5
Four-Stage Thoracoplasty			
8 ribs resected and decortication	1		1
Totals	15	8	23

Six patients, including the three one-stage thoracoplasty patients, required a partial scapulectomy following the resection of five ribs; nine patients had partial scapulectomies after the removal of six ribs; seven patients had a partial scapulectomy after removal of seven ribs; one patient had a partial scapulectomy after the resection of eight ribs.

Of the 23 patients, 15 were given the partial scapulectomy with the last stage, and eight had the partial scapulectomy as a separate procedure.

The patients, both thoracoplasty and scapulectomy, were men ranging in age from 20 to 62 years, with the greatest concentration in the 20 to 29 year age group (37 per cent) and in the 40 to 49 year age group (34 per cent). The average age was 36.8 years.

Goniometric measurements were made on all patients studied, and all but three scapulectomy patients had roentgenograms of the scapulae. To the best of our knowledge all patients had normal shoulder girdle function preoperatively.

SCAPULOHUMERAL RHYTHM

It has been observed roentgenographically[3] that during the movement of abduction of the humerus both the scapula and humerus participate from the start. Except for individual variability during the first 30 degrees of abduction, the relationship between movement at the glenohumeral joint and the scapulothoracic joint remains fairly constant. For each 15 degrees of abduction of the humerus, 10 degrees of motion takes place at the glenohumeral joint and 5 degrees is contributed by the scapulothoracic joint, up to the full excursion of movement to 170 to 180 degrees. Throughout arm abduction the scapula remains in close contact with the chest wall and it rotates outward and upward, the inferior angle moving toward the axilla.

Most of the findings relating to the disturbance of scapulohumeral rhythm were gained from clinical observations.

NATURE OF SURGERY

The thoracoplasties performed at Castle Point entailed a five or six rib resection in two stages or a seven or eight rib resection in three stages, including the removal of the transverse processes. In most cases the posterior approach was used through the usual hockey stick type of incision. The shoulder girdle musculature traumatized during surgery consisted of the middle and lower fibers of the trapezius, the rhomboideus minor and major, the latissimus dorsi and the serratus anterior.

The partial scapulectomiees were performed either in conjunction with the last stage of the thoracoplasty or as a separate procedure (Table I). A variable portion of the distal scapula was resected in a manner to conform to the obliquity of the uppermost intact rib. The muscles which lose part of their scapular attachment are the serratus anterior, the rhomboideus major, the subscapularis, the infraspinatus and the teres major.

SHOULDER GIRDLE CHANGES FOLLOWING THORACOPLASTY

Skeletal Changes.—

After a thoracoplasty there occurs a variable degree of scoliosis, convex toward the operated side in the cervico-dorsal region, and often accompanied by some elevation of the shoulder girdle on the operated side (Fig. 2). However, the shoulder may be at the same level as the opposite side and infrequently it is lower (Fig. 1). Associated with a high shoulder one usually finds a deepening of the supraclavicular fossa.

Seen from a lateral view, an anterior displacement of the shoulder girdle on the operated side is often noted. The depression created by the resection of ribs permits the girdle to move forward into the decostalized area.

On the roentgenogram the scapula on the operated side is usually closer to the vertebral column than is its opposite (Figs. 1, 2). This is due in part to

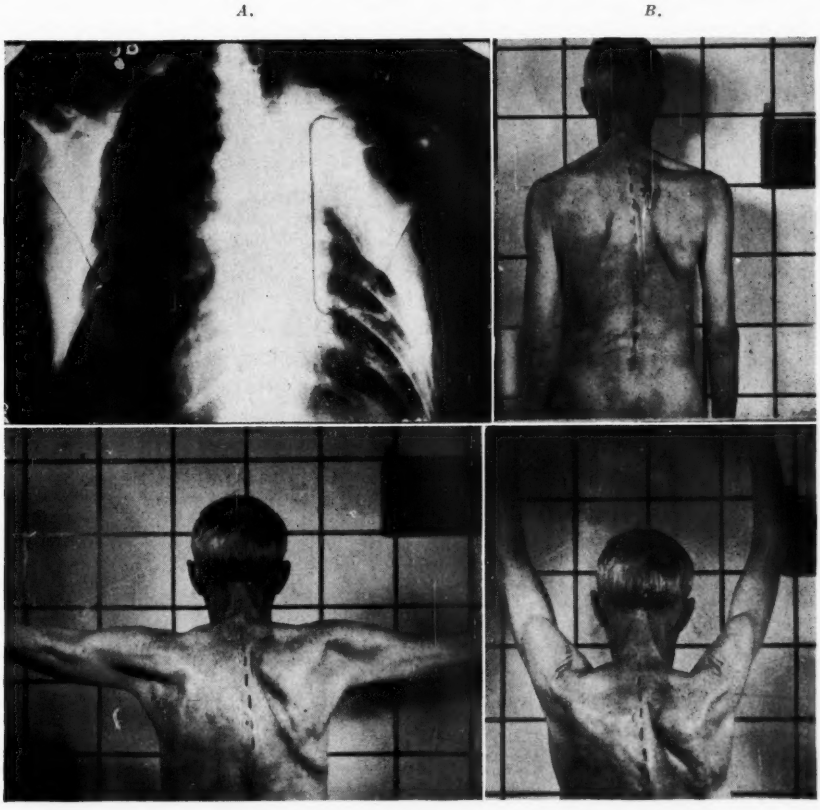


Fig. 1.—A 62-year-old patient who received a two-stage six-rib resection. Roentgenogram shows right shoulder girdle lower than its opposite, and scapula is closer to spinal column. Scapulohumeral angle on left is 37 degrees, on right 36 degrees. *B*, indicates some muscular atrophy in right supraspinous and interscapular areas. *C* and *D* reveal some disturbance in right scapulohumeral rhythm as evidenced by protruding vertebral border and superior angle of scapula, but arm abduction is about equal bilaterally: 156 degrees on left, 159 degrees on right.

a medial displacement of the scapula and in part to the scoliosis with its convexity toward the operated side.

Measurement of the scapulohumeral angle on the operated side of the ten representative thoracoplasty patients showed an insignificant difference from that of the nonoperated side. The difference ranged from minus 1 degree to plus 5 degrees, with an average deviation of plus 2.3 degrees.

A posterior view of the patient often reveals a groove on the operated side between the vertebral border of the scapula and the vertebral column, with some degree of prominence, posteriorly, of the vertebral border of the scapula (Figs. 1, 2). The groove is due to atrophy of the trapezius and of the rhomboids in the line of incision. When sufficient ribs have been removed to allow the whole scapula to sink into the decostalized area, a depression is commonly observed on the upper chest wall, especially in the thin patients, and the vertebral border of the scapula is no longer prominent. The heavy-set patients usually fill in the crests and crevices created by surgery, and their outer bodily contour is less disfigured.

Changes in Muscle Function.—

Experience has shown that, with the exceptions due to complications, the thoracoplasty patients gain normal (Fig. 1) or near normal range—within 15 to 20 degrees—of shoulder girdle movement as compared to the nonoperated side (Fig. 2).

The degree of loss of abduction of the ten representative thoracoplasty patients ranged from 0 to 18 degrees, with an average loss of 5.1 degrees.

Often the limitation of movement, when present, is due to soft tissue resistance, especially of the incised latissimus dorsi, which can usually be overcome with persistent effort.

A common finding following thoracoplasty is a slight disturbance of scapulohumeral rhythm associated with either normal or with some degree of limitation of shoulder girdle movement. This disturbance is evident during humeral abduction, at which time the vertebral border of the scapula is observed to protrude somewhat posteriorly from the chest wall (Figs. 1, 2).

SHOULDER GIRDLE CHANGES FOLLOWING THORACOPLASTY COMBINED WITH PARTIAL SCAPULECTOMY

Skeletal Changes.—

The partial scapulectomies performed at Castle Point consisted of the resection of a variable portion of the distal scapula, at a higher level medially than laterally. On the roentgenogram of the scapulae it was difficult to measure how much of the scapula had been excised because its vertebral border was often obscured by the regenerated ribs.

All of the roentgenograms made of the twenty scapulectomy patients have shown a medial and anterior displacement of the scapula on the chest wall (Figs. 3, 4, 5, 6, 7, 8, 9).

Sixteen of the 20 roentgenograms show some degree of elevation of the shoulder girdle on the operated side (Figs. 3, 8). An anterior view of the patient shows a deepening of the supraclavicular fossa accompanying the elevated girdle.

Seventeen of the twenty roentgenograms present a significant difference between the operated and nonoperated sides in measurements of the scapulohumeral angles, as compared to the roentgenograms of the thoracoplasty patients. The difference in the scapulohumeral angles of each scapulectomy pa-

tient ranged from minus 3 degrees to plus 29 degrees, with an average deviation of 13 degrees, in contrast to the average deviation of 2.3 degrees found in the thoracoplasty group.

The greater scapulohumeral angle on the operated side of the scapulectomy patient indicates that the scapula has rotated inward and what now corresponds to the inferior angle of the scapula has moved closer to the vertebral column.

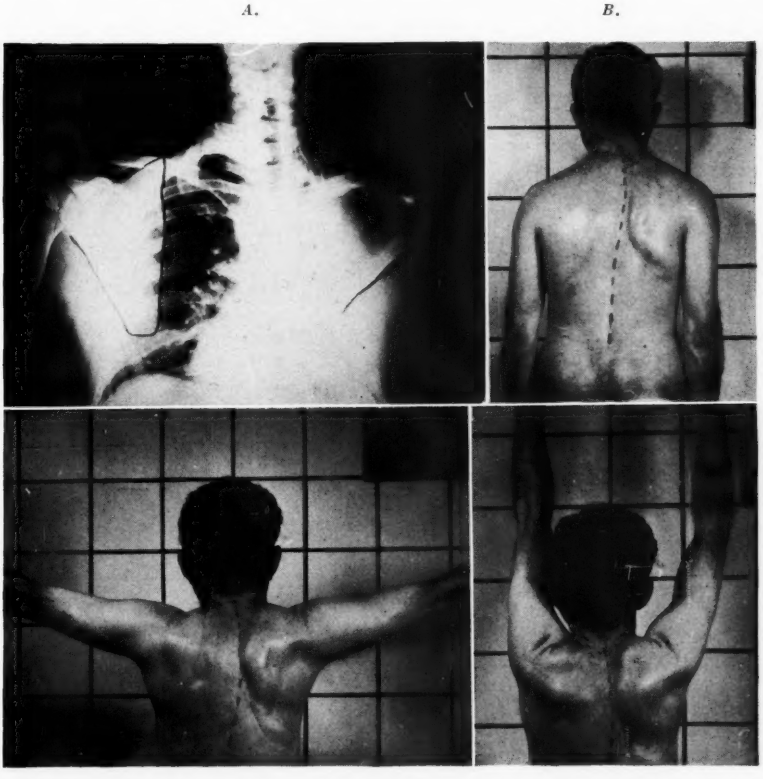


Fig. 2.—A 36-year-old patient who underwent a three-stage, eight-rib resection. Roentgenogram shows right scapula displaced medially, slightly elevated, and rotated inward. Inferior angle and vertebral border are ill-defined. Scapulohumeral angle on left measures 44 degrees, on right 49 degrees. *B* reveals some atrophy in right interscapular area and also posterior prominence of vertebral border of scapula. *C* shows equal arm abduction and scapular rotation bilaterally, with some disturbance of scapulohumeral rhythm on right, as indicated by protruding vertebral border of scapula. Arm abduction in *D* gives a reading of 180 degrees on left, 162 degrees on right.

A posterior view of the scapulectomy patient may or may not show a flaring of the vertebral border of the scapula. The thin patients, more often than the heavy-set patients, display a depression on the upper chest wall where the scapula has been displaced anteriorly into the decostalized space (Figs. 3, 4, 8, 9).

Changes in Muscle Function.—

When abduction of the arms is initiated, a common finding on the operated side is a marked disturbance of scapulohumeral rhythm, characterized by a posterior protrusion of the vertebral border of the scapula with the scapula as a whole moving upward on the chest wall. As movement of both arms continues to a right angle, a lag of scapular rotation on the operated side is noticeable, more apparent in those with greater restriction of motion (Figs. 3, 4). When the patient is asked to stop the movement at shoulder height, although the

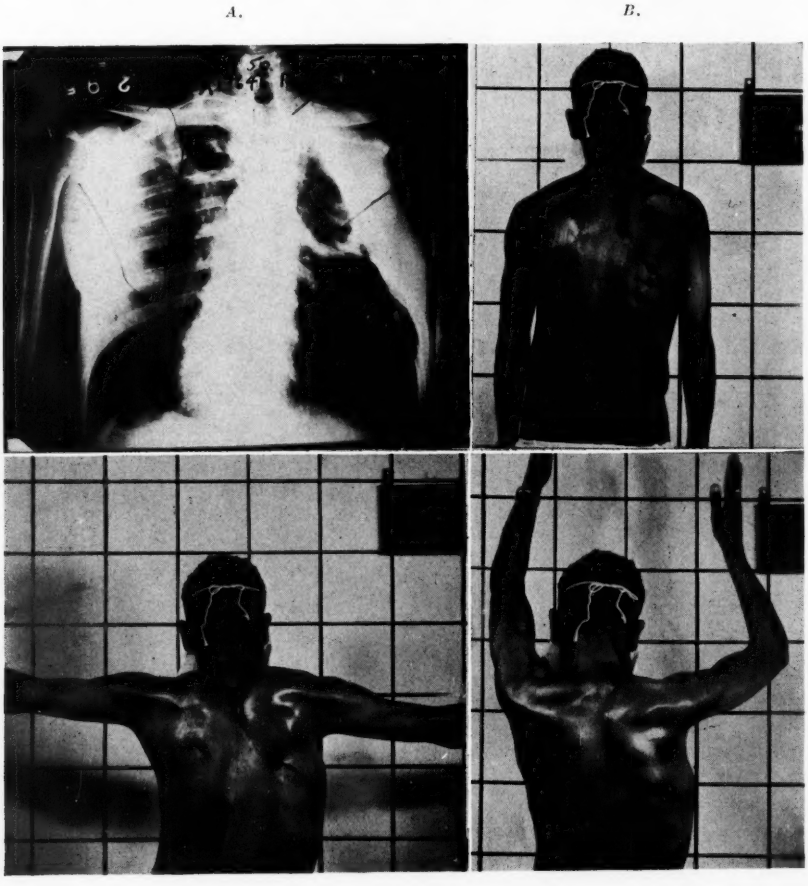


Fig. 3.—A 42-year-old patient with a two-stage, six-rib resection and a partial scapulectomy performed with the second stage. Roentgenogram shows right scapula displaced anteromedially and rotated inward, with the superior angle elevated. Left scapulohumeral angle is 40 degrees, right is 58 degrees. Right upper chest wall in *B* presents a depression where scapula has moved anteriorly into decostalized area. With arms abducted to 90 degrees there is a lag in scapular rotation, right, and arm is at a lower level than its opposite. Also a prominence of right vertebral border of scapula may be noted. Range of full abduction in *D* is 170 degrees on left, 119 degrees on right. Right arm has moved somewhat anteriorly toward flexion.

operated scapula is higher than its opposite member, the arm as a whole is quite perceptibly at a lower level (Figs. 3, 4). Undoubtedly, when this occurs, the surgical damage to the scapular rotators is the cause; this will be discussed later.

As abduction continues beyond 90 degrees, in these patients, the arm of the operated side moves anteriorly toward flexion, the patient being unable to stabilize the shoulder girdle in the position of humeral abduction (Fig. 3).

When the scapulectomy patient has reached the maximum possible range of abduction, and if limitation of movement is present, the extremity can usually be moved passively to the full range but the patient is unable to maintain it there without assistance. The extremity tires easily in the elevated po-

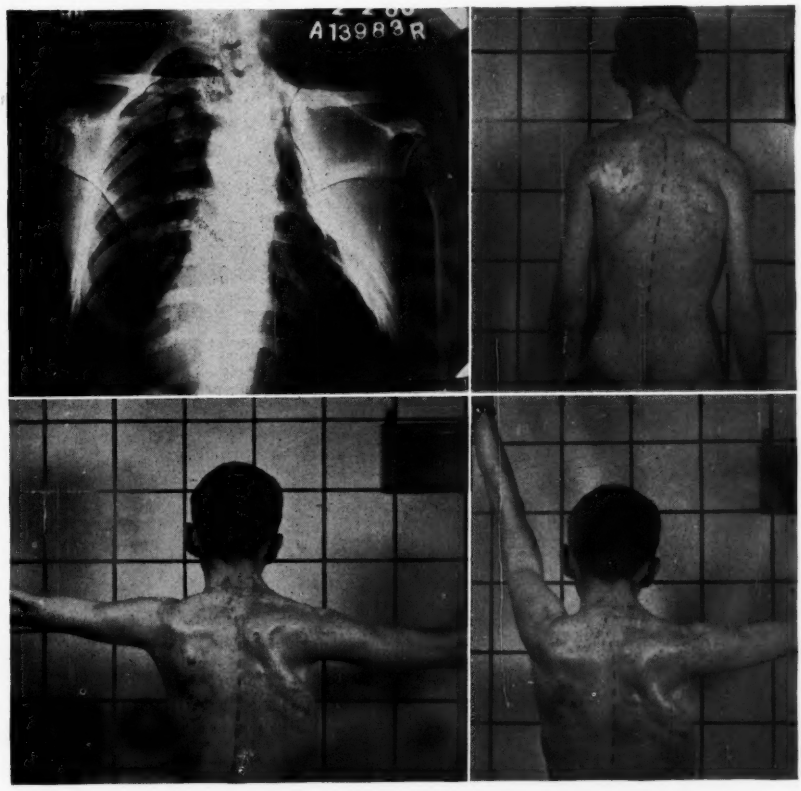
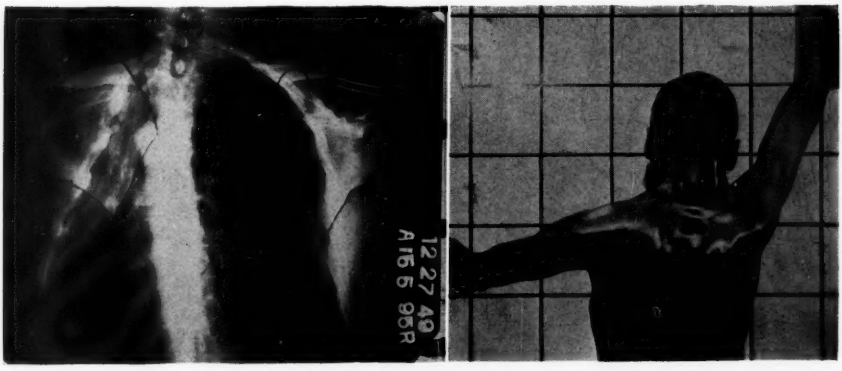


Fig. 4.—A 23-year-old patient who had five and one-half ribs resected in two stages, and a partial scapulectomy during the second stage. Roentgenogram shows right scapula displaced anteromedially and rotated inwardly, with the superior angle elevated. Left scapulohumeral angle measures 54 degrees, the right 83 degrees. *B* shows depression on right upper chest wall where scapula has been displaced anteriorly. At 90 degrees of arm abduction the operated shoulder girdle presents a prominence of the vertebral border and a lag in scapular rotation. Right scapula is elevated and the arm is at a lower level than its opposite. Arm abduction on left is 163 degrees, on right 100 degrees.

sition and must be brought down to rest. Even after six to eight weeks of daily physical therapy the attainment of active full range of shoulder girdle motion is not possible due to the limitation imposed by the surgical procedure.

Sixteen of the twenty-three scapulectomy patients showed a significant difference in the degree of abduction loss when compared to that found in the thoracoplasty group. The amount of restriction for the whole scapulectomy group ranged from 2 degrees to 85 degrees, with an average loss of 35.7 degrees. The thoracoplasty patients displayed an average loss of 5.1 degrees.



A. *B.*

Fig. 5.—A 44-year-old patient who had a three-stage, seven-rib resection and a partial scapulectomy in conjunction with the last stage. Left scapula is displaced anteromedially, rotated inward, and the superior angle elevated, as seen on the roentgenogram. Scapulohumeral angle on left is 63 degrees, on right 42 degrees. In *B* the arms are abducted to their limit. Left scapula is elevated and the vertebral border protrudes posteriorly. Abduction on left is 72 degrees, on right 157 degrees.

There appeared to be no consistent relationship between the scapulohumeral angle and the degree of abduction loss on the operated side. For instance, the patient with the greatest difference between his two scapulohumeral angles, that of 29 degrees, displayed an abduction loss of 63 degrees (Fig. 4). The patient with the greatest loss of abduction, 85 degrees, showed a 21 degree difference in scapulohumeral angles (Fig. 5). The patient with an 81 degree loss of abduction gave a scapulohumeral angle difference of only 11 degrees (Fig. 6). Still another patient with a 7 degree loss of abduction had a difference of 16 degrees in scapulohumeral angles (Fig. 7).

The question arises: "What are the factors that cause the increase in scapulohumeral angle and the degree of restriction of arm abduction?"

DISCUSSION

The disturbance of scapulohumeral rhythm observed on the scapulectomy patients is intimately related to the increase of the scapulohumeral angle and to the degree of restriction of humeral abduction. Let us examine closely the muscular dysfunction underlying these changes.

Scapular rotation during arm abduction normally elicits contraction from two groups of muscles, an upper and a lower group.[3] The upper component, consisting of the levator scapula, the upper fibers of the trapezius, and the upper portion of the serratus anterior, is not nearly as much disturbed during thoracoplasty and partial scapulectomy as is the lower component; i.e., the lower fibers of the trapezius and the lower digitations of the serratus anterior.

A. *B.*

Fig. 6.—A 58-year-old patient who underwent a three-stage, seven-rib resection and a partial scapulectomy with the last stage. Roentgenogram displays an anteromedial displacement and inward rotation of left scapula. Scapulohumeral angle on left is 45 degrees, on right 34 degrees. In *B* left scapula is elevated and the vertebral border protrudes posteriorly. Abduction is limited to 74 degrees on left, 155 degrees on right.

A. *B.*

Fig. 7.—A 23-year-old patient who had a two-stage, six-rib thoracoplasty and a partial scapulectomy with the second stage. Roentgenogram shows an anteromedial displacement and inward rotation of left scapula. Left scapulohumeral angle measures 63 degrees, the right 47 degrees. Range of arm abduction on left is 166 degrees, on right 173 degrees. Some prominence of left vertebral border of scapula is evident.

Trapezius and Serratus Anterior.—

The relationship between the trapezius and the serratus anterior in shoulder girdle movements is a most interesting one. Normally the two muscles act simultaneously to rotate the scapula during abduction of the humerus. When the trapezius is paralyzed, as in a spinal accessory nerve lesion, the serratus anterior alone is capable of rotating the scapula completely on the thorax and thereby permitting full range of abduction.[4-6] However, when the serratus anterior is completely involved, as in a long thoracic nerve lesion, the trapezius alone is incapable of rotating the scapula, and the humerus cannot be abducted much beyond a right angle.[4-8]

Normal scapulohumeral rhythm requires the coordinated effort of all the shoulder girdle muscles. However, if one may designate the relative importance of the various shoulder girdle muscles, the most important one, in the authors' estimation, is the serratus anterior. Without its function the shoulder girdle suffers greater disability than when any other single muscle is paralyzed.[6]

Scapulohumeral Angle.—

Particular attention is drawn to the function of the serratus anterior since this seems to be such a significant factor in the cause of the increased scapulohumeral angle and the limitation of abduction following partial scapulectomy.

Spalteholtz[9] notes that, of the three portions of the serratus anterior, the lowest portion, which includes the fourth to tenth digitations and converges to the inferior angle of the scapula, is the strongest.

Special cognizance is taken of the fact that the greatest concentration of the serratus anterior fibers occurs at the inferior angle of the scapula, and it is precisely this area that is excised during a partial scapulectomy.

Here is a good reason for the increase in the scapulohumeral angle after a partial scapulectomy. Normally, at rest, the serratus anterior maintains the scapula close to the chest wall and its vertebral border parallel to the spinal column. The loss of function of the lower fibers of the serratus anterior causes the scapula to rotate inward, the inferior portion moving closer to the vertebral column, with the angle between the humerus and the lateral border of the scapula (the scapulohumeral angle) thereby increased. On the roentgenogram the superior angle of the scapula is higher than its opposite (Figs. 3, 4, 5).

Probably the degree of inward rotation of the scapula is influenced by the amount of the distal scapula resected and by the manner of resection. The patient with the greatest increase of scapulohumeral angle appears to have had more than the usual amount of the distal scapula resected (Fig. 4). Those who showed a lesser increase of the scapulohumeral angle probably had less of the scapula resected. It was not always possible to distinguish on the roentgenogram how much was resected.

Of those scapulectomy patients who displayed no change or an insignificant change in the scapulohumeral angle, the manner of resection may have been modified. This was definitely the case in an operation observed by one of the writers (H. B. D.). The inferior angle and the medial and lateral borders adjacent to it were "trimmed" rather than resected in the usual manner. On the

roentgenogram (Fig. 8) the scapula appears somewhat shorter than its opposite member but it retains something of its original configuration. In this instance, with the vertebral border relatively little disturbed and with less of the inferior angle removed, not only did the scapulohumeral angle show little change (3 degrees) but arm abduction showed a correspondingly negligible loss (5 degrees).

Degree of Abduction.—

Without scapular rotation the complete range of arm abduction is not possible. Scapular rotation and arm abduction will be interfered with to the extent that the lower fibers of the serratus anterior lose their area of insertion and with it their effective leverage on the scapula. Furthermore, the lower

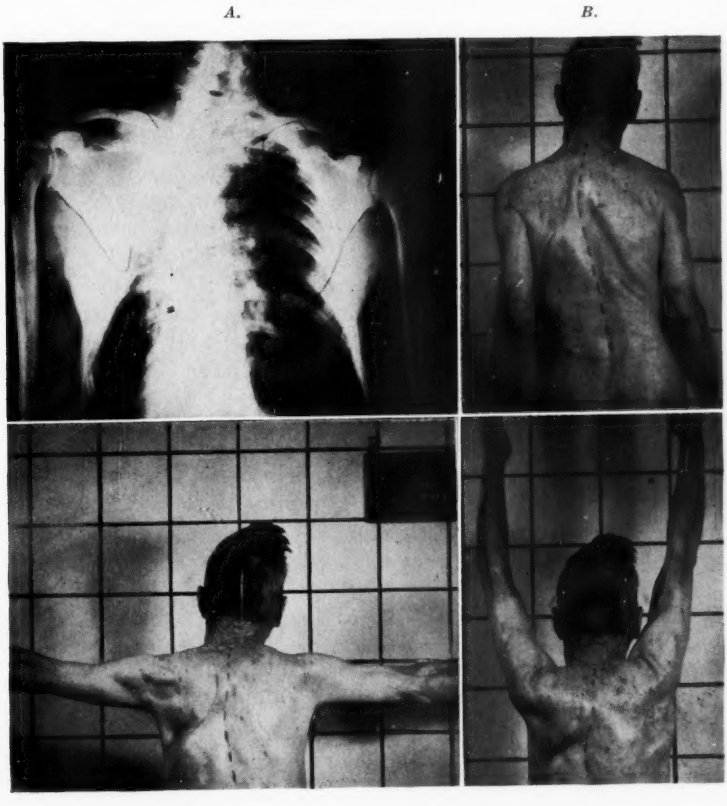


Fig. 8.—A 45-year-old patient who had a three-stage, seven-rib thoracoplasty at another institution, and a partial scapulectomy at Castle Point. Roentgenogram reveals an anteromedial displacement and elevation of left scapula. Scapulohumeral angle on left is 36 degrees, on right 33 degrees. *B* shows depression in left upper chest wall where scapula has moved anteriorly into decostalized area. In *C* the scapulae and arms appear to be almost symmetrical in position, with a slight disturbance of scapulohumeral rhythm (left vertebral border of scapula protrudes posteriorly). Full arm abduction gives a reading of 161 degrees on left and 166 degrees on right. Difference in range of abduction appears to be greater than actually due to deviation of head and neck to right.

fibers of the trapezius are weakened by the thoracoplasty and this in turn makes its contribution to the shoulder girdle dysfunction.

Involvement of the serratus anterior and of the lower trapezius manifests itself during arm abduction as a disturbance of scapulohumeral rhythm, the scapula moving upward on the chest wall and its vertebral border protruding posteriorly.

It has already been mentioned that there was no consistent relationship between the increase of scapulohumeral angle and the degree of abduction limitation. Occasionally some cause for diminished abduction may lie in the personality of the patient. Such a personality may be described, characteristically, as tense, apprehensive, and intolerant of pain.

The concept of the mechanism at work in this type of patient is that the surgical trauma, induced on a musculature already hypertonic, tends to increase the existing muscular tension. In addition, the low tolerance for pain discourages the patient from moving the extremity beyond the point of pain, however slight it may be. He tends to protect and favor the operated side. When the arm is moved it is done so reluctantly, the apprehension serving to increase reflexly the tone of the muscles antagonistic to those of the particular movement desired. When it is abduction of the humerus that is desired, the adductors of the humerus—the pectoralis major and the latissimus dorsi—increase in tension.

If this sort of reaction persists for any length of time one may well expect adhesion formation between the scapula and the adjacent tissues on the thoracic wall. Once this occurs the restriction in movement is marked and difficult to overcome. The patients in Figs. 5 and 6 illustrate just this condition.

Other Factors Affecting Shoulder Girdle Function.—

It should be noted as well that trauma to the various shoulder girdle muscles, other than those already discussed, contributes to shoulder girdle dysfunction. When the scapulectomy patients reach their individual limits of abduction, the affected arm often tires easily in that position. This may be due partially to the weakening or loss of action of the teres major in its capacity of assisting the arm to maintain a static position.[3]

Quite often it is observed that during abduction the arm advances toward flexion as it is elevated beyond the right angle (Fig. 3). Interference with the function of the serratus anterior which normally maintains the scapula close to the chest wall and of the rhomboids and the middle fibers of the trapezius which ordinarily fix the scapula in their plane of action prevents proper fixation of the scapula, thereby permitting forward movement on the chest wall during humeral abduction.

The subscapularis and the infraspinatus muscles are somewhat affected by a partial scapulectomy and this, too, probably weakens movement at the glenohumeral joint.

A previous study of thoracoplasty patients[1] indicated that age is not a significant factor in relation to the degree of skeletal deformity or in the changes of shoulder girdle function. Similarly, no relationship between age of patient

and shoulder girdle changes could be found in the comparatively small number of patients included in this study.

The muscular development of the patient before surgery is a more important factor. This is best illustrated by a patient (Fig. 9) who was originally listed as having had a thoracoplasty only. Upon examination the shoulder girdle on the operated side behaved like that of a thoracoplasty patient with normal range of movement. However, the roentgenogram of the scapulae showed a difference of 17 degrees between the two scapulohumeral angles, which is a greater difference than that found within the range of the representative thoracoplasty patients. The roentgenogram further revealed a difference in the outlines of the

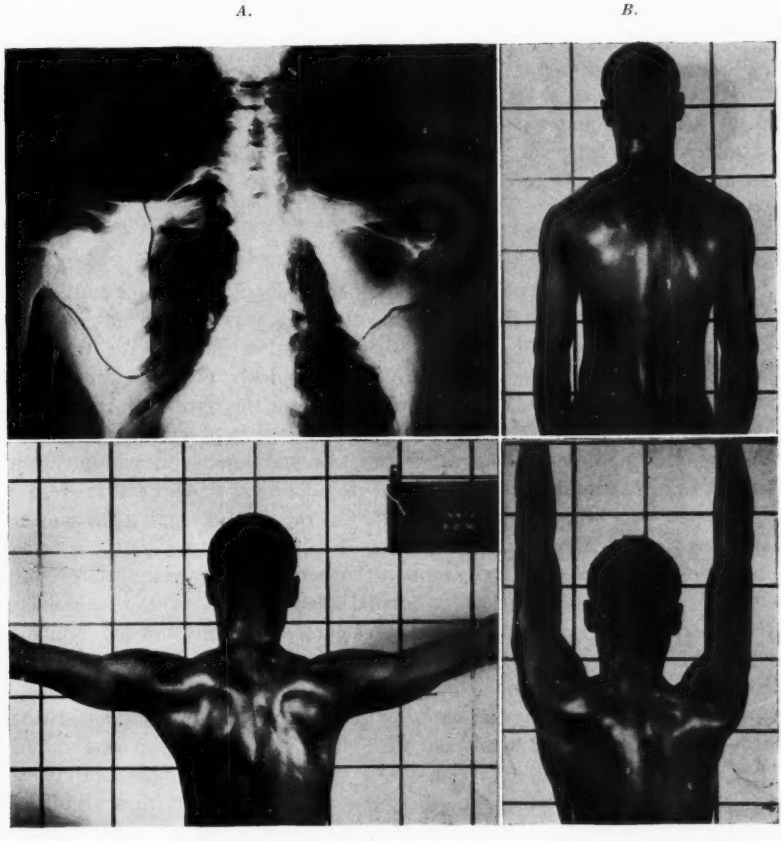


Fig. 9.—A 27-year-old patient who underwent a two-stage, six-rib thoracoplasty and a partial scapulectomy in conjunction with second stage. On roentgenogram right scapula is displaced anteromedially and rotated inward. Scapulohumeral angle on left is 43 degrees, on right 60 degrees. Depression in right upper chest wall is apparent in *B*. Right shoulder girdle is elevated slightly. Abduction of arms in *C* and *D* shows them to be equal and symmetrical, with a slight protrusion of vertebral border of right scapula. Abduction on left is 178 degrees, on right 177 degrees.

two scapulae which made it seem likely that a partial scapulectomy had been performed in conjunction with the second stage of the thoracoplasty. This was confirmed by a review of other roentgenograms taken before and after surgery.

What probably happened in the above-mentioned case was that the amount of scapula resected did not seriously interfere with lower serratus anterior function, and the good strength of the other scapular rotators made normal shoulder girdle function possible. More difficult to explain is the degree of difference in the scapulohumeral angles.

Of those patients in whom a poorly developed musculature may have contributed to shoulder girdle dysfunction, we can only speculate as to the mechanism involved. One may well ask, "Has a wide scar formation occurred in the healing of the incised muscles?" Then, too, the adherence of the scar in the skin and subcutaneous tissue to the scar in the underlying musculature, and the formation of adhesions in the operative field may further complicate the picture. Certainly these and the other factors already discussed, such as the surgical procedure and the personality of the patient, influence the end-result of shoulder girdle function. Each patient presents one or more of these factors in various combinations and it is not often possible to say with assurance which causative factors determine the end-result.

SUMMARY AND CONCLUSIONS

1. During a two-year period the shoulder girdle dysfunction resulting from combined thoracoplasty and partial scapulectomy was studied on a group of twenty-three patients in a Veterans Administration hospital for tuberculous patients; in addition, ten representative thoracoplasty patients were similarly studied to observe the shoulder girdle changes after this type of surgery.

2. Roentgenograms of the scapulae were utilized to observe skeletal changes of the shoulder girdle on the operated side, and goniometric measurements were taken to record shoulder girdle movements. Clinical examinations were performed to note the disturbance of scapulohumeral rhythm during the movement of abduction of the humerus.

3. Those patients who underwent a thoracoplasty showed certain changes in skeletal relationship: an anteromedial displacement of the scapula, usually some degree of shoulder girdle elevation, and slight inward rotation of the scapula, which increased the scapulohumeral angle; i.e., the angle formed between the long axis of the humerus and the axillary border of the scapula. Changes in muscle function in these patients consisted of a mild disturbance of scapulohumeral rhythm associated with either no limitation of arm abduction or with limitation of slight degree—15 to 20 degrees. The scapulectomy patients, on the other hand, showed the same changes but to a significantly greater extent, especially in the increase of scapulohumeral angle and in the restriction of abduction of the humerus.

4. The role of the serratus anterior in shoulder girdle mechanics is stressed. This muscle, so essential for scapular rotation and complete arm abduction, should be disturbed as little as possible during surgery if good shoulder girdle function is to be preserved.

5. The discussion is centered around the factors which cause the disturbance of the scapulohumeral rhythm, the increased scapulohumeral angle, and the limitation of shoulder girdle function. Among the factors discussed are the shoulder girdle muscles affected by surgery, the patient's personality, the formation of adhesions in the operative field, the age of the patient, and the strength of the shoulder girdle muscles prior to surgery.

REFERENCES

1. Goldberg, J., Friedlander, R., Doppelt, H. B., and Miller, D. E.: Physical Therapy in Post-Thoracoplasty, Am. Rev. Tubere. **60:** 189-205, 1949.
2. Codman, E. A.: The Shoulder. Rupture of the Supraspinatus Tendon and Other Lesions in or About the Subacromial Bursa, Boston, 1934, privately printed.
3. Inman, Verne T., Saunders, J. B. De C. M., and Abbott, Leroy C.: Observations on the Function of the Shoulder Joint, J. Bone & Joint Surg. **25:** 1-30, 1944.
4. Brunnstrom, Signe: Muscle Testing Around the Shoulder Girdle, J. Bone & Joint Surg. **23:** 263-272, 1941.
5. Brunnstrom, Signe: Comparative Strength of Muscles with Similar Function, Physio. Rev. **26:** 59-65, 1946.
6. Wright, Wilhelmine: Muscle Function, New York, 1928, Paul B. Hoeber, Inc.
7. Thompson, George C. V.: Paralysis of the Serratus Anterior Muscle Complicating Dislocation of the Shoulder, M. J. Australia **1:** 231-232, 1941.
8. Griffin, J. P.: Isolated Paralysis of the Serratus Anterior Muscle, J. Indiana State M. A. **42:** 1068-69, 1949.
9. Spalteholtz, Werner: Hand Atlas of Human Anatomy, ed. 7 in English, One-Volume Edition, Philadelphia, 1943, J. B. Lippincott Co.

PULMONARY DECORTICATION FOR CLOTTED HEMOTHORAX FOLLOWING IDIOPATHIC SPONTANEOUS HEMOPNEUMOTHORAX

GABRIEL P. SELEY, M.D., AND HAROLD NEUHOF, M.D.
NEW YORK, N. Y.

THE treatment of clotted hemothorax by pulmonary decortication has become an accepted and often practiced procedure since World War II. Williams[3] reports a case of spontaneous pneumothorax with hemorrhage treated by decortication in 1947, and mentions a similar case reported by Elrod and Murphy[1] in which the patient was operated upon also in 1947. These latter authors[1] believe that their case is the first of this type to have been subjected to decortication. In a presentation on Feb. 18, 1949, before the New York Society for Thoracic Surgery, one of us (GPS) briefly mentioned a case of a patient with spontaneous hemopneumothorax treated by pulmonary decortication by Dr. Harold Neuhof in 1936, and also included a similar case of a patient operated upon by Dr. A. S. W. Touroff on Feb. 17, 1946. In order to attempt to establish priority for the procedure of decortication in the treatment of clotted hemothorax following spontaneous hemopneumothorax, a detailed report of the first case is indicated.

History.—R. T., No. 393576. A 23-year-old white man was admitted to The Mount Sinai Hospital on May 23, 1936, for the treatment of a collapsed right lung. Five and one-half weeks prior to admission, while at work as a clerk, he experienced sudden severe needle-like pain commencing in the right chest anteriorly just lateral to the sternum above the nipple and radiating straight through posteriorly. Pain persisted, but there was no immediate cough, dyspnea, fever, or hemoptysis. The following day a physician was consulted because of persistent pain, slight dyspnea, and vomiting. A thoracentesis was performed, and blood was removed from the right hemithorax. The patient was told that his lung was collapsed. He was admitted to another institution and repeated thoracenteses revealed old blood and serum. Roentgenograms showed complete collapse of the right lung with air and fluid in the right chest. Dyspnea decreased, but he developed a nonproductive painless cough. Pain disappeared and did not recur.

Physical Examination.—Trachea appeared in the midline, entire right chest moved only slightly with respirations, ribs were retracted, diminished fremitus was present anteriorly, tympany was noted anteriorly with moderate diminution of breath sounds posteriorly except at the base where there was flatness and absent fremitus and breath sounds.

Laboratory Data.—Hemoglobin 78 per cent, white blood cells 12,400, polymorphonuclears 79, lymphocytes 16, monocytes 4, eosinophiles 1. Sedimentation rate one hour and twenty minutes (for 18 mm. drop).

Roentgenogram on May 25, 1936, showed fluid and air in the right chest with a fluid level at the fourth rib anteriorly, the heart and mediastinum were drawn to the right, and the right lung was collapsed.

Discussion.—Although the bleeding had stopped, it was decided to explore the chest by open thoracotomy in order to attempt to release the collapsed lung and restore it to normal function. Since this was without precedent a great deal of discussion followed, and many arguments were advanced in favor of the conservative method of treatment by aspiration and watchful waiting. A review of the literature at that time failed to reveal any similar attempts of treatment by open thoracotomy to release the bound down lung. In fact, many authors were of the opinion that blood in the pleural cavity did not clot, but remained liquid

Received for publication Jan. 29, 1951.

and therefore was always absorbed without the formation of clot, restricting membrane, and resultant collapsed lung. Other authors claimed that the blood was defibrinated by the churning action of the heart, and the resultant fibrin might result in pleurisy and adhesions. As a result of these conflicting ideas, treatment consisted of either bed rest or aspiration of blood and replacement by air. However, there were a number of autopsy reports showing collapsed lungs as a result of previous hemopneumothorax with membrane formation. We had had similar experiences in cases which had permanently collapsed lungs following hemorrhage into the pleural cavity. In fact, at the time there was a patient on the same ward who had developed a total cholesterothorax following a steering wheel injury to the chest many years prior to admission, and he required a complete Schede thoracoplasty following evacuation of the cholesterothorax. There had also been some experimental work in animals to show that blood clotted in the closed thorax even more rapidly than outside the chest. The argument was advanced, therefore, that in view of the fact that blood clots in the thorax, a restricting membrane can and does form in certain cases, that permanent crippling of the lung can be produced by this membrane, at least some attempt should be made to avoid this chain of events by early thoracotomy and removal of the restricting membrane.

Operation.—May 23, 1936.—*Anesthesia:* avertin per rectum followed by gas-oxygen-ether by inhalation. A posterolateral incision was made in the seventh right intercostal space extending into the anterior axilla. Old blood clot which was present chiefly in the costophrenic sinus was removed and the field inspected. The collapsed lung was completely enveloped by a brownish homogeneous membrane extending from the prevertebral region to the anterior mediastinal area. This membrane was removed, and the lobes completely released. When the lung was inflated, it appeared perfectly normal. No obvious source for the bleeding was visualized, but it was assumed that it came from the lung. Layer closure was performed without drainage. Complete inflation of the lung was maintained as the thorax was closed.

Course.—Postoperative course was entirely uneventful. Roentgenogram on May 27, 1936, one day postoperative, showed almost complete reexpansion of the lung. On June 4, 1936, there was complete reexpansion of the lung. Patient was discharged well on the eleventh postoperative day.

FOLLOW-UP examinations and roentgenograms over a period of four years showed a completely expanded normal lung and the patient remained symptom free.

SUMMARY AND CONCLUSIONS

1. A detailed case report of a successful pulmonary decortication for clotted hemothorax following spontaneous hemopneumothorax is herein presented.

2. Attention is directed to the fact that this procedure was performed almost fifteen years ago, and probably represents the first operation of this type for spontaneous clotted hemothorax.

3. The rationale for the procedure and pertinent discussion is included.

REFERENCES

1. Elrod, Parker D., and Murphy, James D.: Spontaneous Hemopneumothorax Treated by Decortication, J. THORACIC SURG. **17**: 401, 1948.
2. Touroff, A. S. W., and Seley, G. P.: Pulmonary Decortication in Civilian Practice, J. Mt. Sinai Hosp. **17**: 523, 1951.
3. Williams, Mark H.: The Technique of Pulmonary Decortication and Pleurolysis, J. THORACIC SURG. **20**: 652, 1950.

POSTERIOR MEDIASTINAL TRACHEOTOMY

HERBERT SLOAN, M.D., AND R. ADAMS COWLEY, M.D.
ANN ARBOR, MICH.

CASE REPORT

TRACHEAL obstruction at a level so low that it cannot be relieved by the usual cervical tracheotomy presents a difficult therapeutic problem. This is particularly true when the obstruction results from compression of the trachea by an aneurysm. The introduction through a cervical incision of a tracheotomy tube sufficiently long to pass beyond the obstruction is almost certain to be followed by erosion of the aneurysm and its rupture into the trachea. Renewed interest in the treatment of such aneurysms by polythene wrapping or wiring has once more emphasized tracheal obstruction as a complication of aortic aneurysm.[1-3]

More than forty years ago the question of low tracheal obstruction was extensively discussed and several solutions to it were suggested. In 1907 Gluck[4] proposed posterior bronchotomy by an extrapleural approach through the posterior mediastinum as a means of relieving this type of tracheal obstruction. It is not apparent that he ever carried out the procedure.[5] Other investigators decried this approach and felt it violated fundamental principles of thoracic surgery.[6, 7] Instead it was believed the solution lay in the formation of a lung or bronchial fistula[6-9] which, we understand, was intended to mean (1) an incision into the pulmonary substance to such an extent that a bronchus or bronchi of sufficient caliber for retrograde respiration would be opened; (2) the amputation of a lobe or portion of a lobe sufficient to accomplish the same purpose; (3) the transpleural construction of a bronchial fistula by an attempt to suture skin flaps directly to an incision in the bronchial wall. Kuttner[10] in 1908 first attempted to produce a bronchial fistula in a patient with compression of the trachea from an aortic aneurysm. An upper lobe was resected and the upper lobe bronchus fixed in the wound. This patient died a few hours postoperatively. In 1922 Rethi[7] reported the formation of a lung fistula by means of a two-stage operation in a patient with tracheal obstruction from an aneurysm. The patient lived fourteen months, twelve days, and respiratory exchange through the fistula was adequate. It was not until 1929 that an attempt was made by Jirasek[11] to perform a posterior bronchotomy as originally advocated by Gluck. Unfortunately this patient died the day following operation.

Attempts to relieve respiratory obstruction by posterior bronchotomy or the construction of a lung or bronchial fistula have been uncommon and, except for Rethi's case, almost uniformly unsuccessful. It has been more

From the Department of Surgery, University of Michigan Medical School.
Received for publication Feb. 12, 1951.

common to attempt to relieve tracheal obstruction by decompressive mediastinotomy as was suggested by Guleke,[12] and more recently re-emphasized by Abbott.[2] As Abbott points out, however, several weeks must elapse following decompression of the mediastinum before the full benefits of the operation are obtained. In our patient, a 48-year-old man with severe respiratory obstruction from tracheal compression by a large aortic aneurysm, the severity and steady progression of the respiratory obstruction and the peculiar deformation of the trachea by the posteriorly projecting aneurysm made it seem unlikely that mediastinal decompression would restore unobstructed respiration. Instead, we chose to attempt the introduction of a tube into the trachea below the level of obstruction through an extrapleural approach in the posterior mediastinum. The proposed procedure was a modification of that suggested by Gluck many years ago.

CASE REPORT

History: J. N., U. H. No. 679024, a 48-year-old white man, was admitted to the Medical Service of the University Hospital on April 4, 1950, complaining of progressive difficulty in breathing. For several years the patient had had frequent winter respiratory infections which had increased steadily in severity. During these episodes his cough was productive of greenish-yellow sputum, and he was slightly dyspneic.

In October, 1949, the patient had an exacerbation of this illness. His cough became productive of three cups of greenish, thick sputum each day. He had continued fever and grew progressively more dyspneic. Respirations were labored and accompanied by wheezing. The patient had been hospitalized elsewhere at this time for eight days, receiving oxygen, penicillin, and streptomycin. He was discharged slightly improved, but his symptoms continued. There had been a twenty-five pound weight loss. He denied venereal disease.

Physical Examination: Temperature was 100.2° F.; blood pressure, 140/58; pulse, 92; respiration, 20 (labored).

On examination the patient was acutely ill and seriously dyspneic. There was an obvious expiratory tracheal wheeze. Expirations were prolonged and forced. Râles were heard over each lower lung field. Retromanubrial dullness was increased. A tracheal tug was felt. On auscultation of the heart there was a probable aortic diastolic murmur heard over the aortic area and along the left border of the sternum. A systolic murmur was present in the aortic area. The pupils were small and fixed. There was clubbing of the fingers. The patient was producing about 40 to 180 Gm. of purulent sputum each day. He was markedly cyanotic during frequent coughing attacks.

Laboratory Data: Kahn, 4 plus; hemoglobin, 14.0 Gm.; red blood cells, 4.73 million; white blood cells, 16,800; urine, normal findings.

Roentgenograms (which are not sufficiently clear for reproduction): Examination of the chest showed an aneurysm of the ascending, transverse, and descending aorta with a posterior dilatation which pushed the trachea posteriorly and to the right, greatly compressing it. There was extensive calcification in the aneurysmal wall. Angiocardiograms confirmed these findings but did not establish definitely the points of origin of the great vessels with respect to the aneurysm. Changes present in the right base were suspected of being pneumonitis or bronchiectasis. There was no evidence of vertebral erosion.

Course: Tracheoscopy was performed May 1, 1950. The trachea was reduced to a narrow slit at a distance of 20 centimeters from the upper gums by a mass projecting into the left tracheal wall. No pulsations of the mass were noted. On expiration the tracheal lumen was closed almost completely. Large quantities of brownish-green purulent secretions were coughed up during the procedure. The patient was given penicillin intramuscularly 400,000 units twice daily, and penicillin aerosol three times daily. The amount of purulent sputum decreased. Streptomycin, 0.5 Gm. intramuscularly twice daily, was added later.

On May 9, 1950, the patient was transferred to the Thoracic Surgery Service when it became apparent that the symptoms of respiratory obstruction were increasing steadily in severity. On May 22, 1950, the right posterior mediastinum was explored and a posterior tracheotomy carried out. Following local anesthesia of the pharynx a small endotracheal tube was introduced past the major point of tracheal obstruction. The patient was then anesthetized with ether-oxygen in a closed system. Through a vertical incision paravertebral segments, six to eight centimeters in length, of the third, fourth, and fifth ribs were removed. Extrapleural dissection was carried out, and the trachea, which deviated posteriorly and to the right, was exposed. The markedly compressed trachea was partially imbedded in the aneurysm. A longitudinal incision was made in the posterior membranous wall of the trachea approximately four centimeters above the carina. A number of Portex endotracheal tubes had been readied for introduction into the trachea but none could be sufficiently angulated to keep it in place. The surgeon was finally obliged to use a right-angled 38 French Pezzer catheter after removing its tip three centimeters beyond its angle. This was introduced into the trachea with the open end pointing toward the main carina.

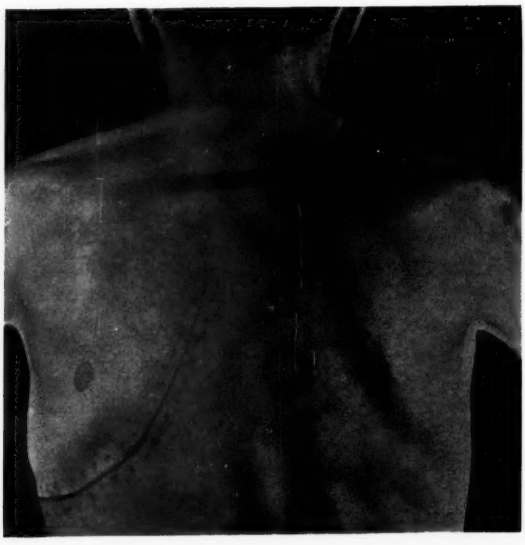


Fig. 1.—Photograph of the patient taken following insertion of the tracheotomy tube and subsequent left thoracotomy with partial wrapping of the aortic aneurysm.

The wound was closed about the catheter after it had been tacked to the prevertebral fascia and the fascia of the erector spinae muscles with a few interrupted sutures of 3-0 silk. A Penrose drain was placed down to the area of the tracheal incision. The small endotracheal tube that had been used during the operation was removed. Satisfactory respiratory exchange occurred through the tracheotomy tube. Following operation the patient was no longer dyspneic, and all respiratory distress disappeared.

Postoperatively there was no evidence of mediastinal infection or emphysema. It was possible to keep the transthoracic tracheotomy tube and bronchial tree free of secretion by means of a small catheter which could be passed beyond the angle of the tracheotomy tube. Constant "cold steam" was administered in conjunction with oxygen. After a few days 2 c.c. quantities of warm isotonic saline solution containing 80,000 units of penicillin were employed to irrigate the tube at from one- to three-hour intervals. The bronchial secretions,

which had been so profuse immediately following operation, diminished progressively in amount and became less purulent in character.

On May 29, 1950, a left thoracotomy was carried out and the transverse and descending portions of the arch of the aorta were wrapped with strips of polythene film.* There was diffuse dilatation of the entire visible arch of the aorta. The descending aorta was also dilated. No definite involvement of the left carotid artery or left subclavian artery was

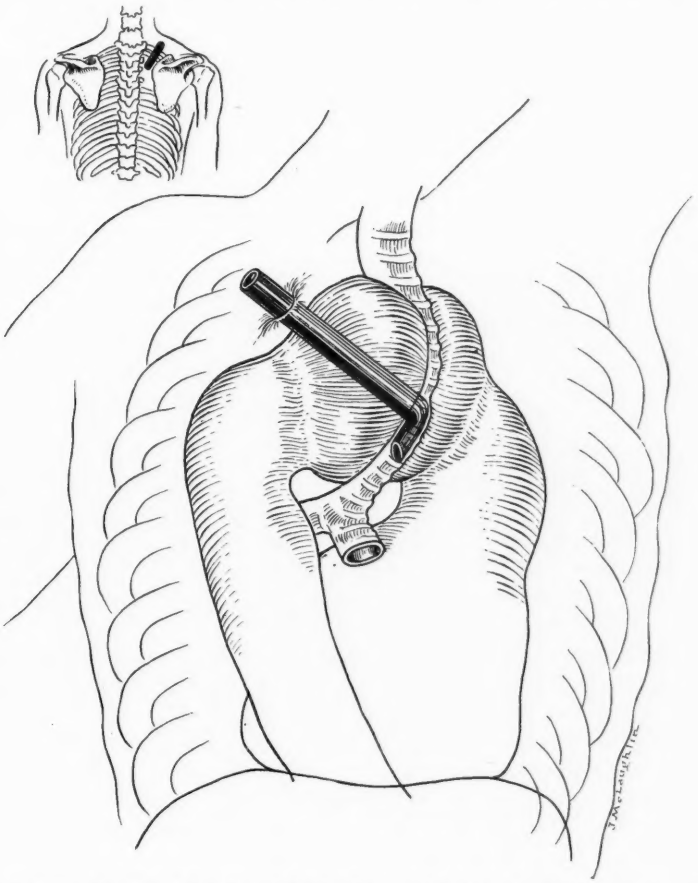


Fig. 2.—Diagram showing deformity of the trachea produced by the aortic aneurysm and the point of insertion of the tracheotomy tube.

present. To the right of the vertebral column a secondary posteriorly projecting portion of the aneurysm was found.

The patient did well postoperatively on the same regimen, but on June 13, 1950, it was necessary to change the tracheotomy tube which had become plugged with dried secretions. A new tube, similar to the original, was reintroduced into the trachea through the back by threading it over a four millimeter bronchoscope and guiding the tube into the trachea under direct vision. Thereafter, no further obstruction of the tracheotomy tube occurred.

*1.5 mil. solvent cast DuPont Polythene, NV-7-14, furnished through the courtesy of E.I. duPont de Nemours and Company, Wilmington, Delaware.

Subsequent to this, on July 3, 1950, and on July 7, 1950, attempts were made to remove the tracheotomy tube permanently, but it became apparent that it had been inserted very near to the point of greatest obstruction of the trachea and that the left-sided wrapping of the aneurysm had not as yet significantly decreased the aneurysm's pressure against the trachea.

On July 21, 1950, a right thoracotomy was performed and that portion of the aneurysm visible in the right chest was wrapped with polythene film. The flattened, compressed trachea, which had been pushed far to the right and posteriorly, lay partially imbedded in the thin, pulsating wall of the aneurysm. The ascending portion of the arch of the aorta was greatly dilated, and it expanded posteriorly as a secondary aneurysm which involved the trachea. The innominate artery did not appear to be involved. Dissection was carried medially to the limits of the previous wrapping. The trachea was completely freed from the aneurysm.

The patient again recovered satisfactorily. He maintained a weight gain of twenty-two pounds and had no respiratory distress. Suctioning was required infrequently, and the patient no longer needed "cold steam." He became ambulatory and felt well for the first time in many months.

On Aug. 22, 1950, the posterior tracheotomy tube was permanently removed. The trachea was found on bronchoscopy to be widely patent, although there were granulations about the tracheotomy wound. The patient was able to breathe normally when the tube track was occluded.

On Sept. 1, 1950, bronchoscopy was again carried out. The healing tracheotomy wound was seen with difficulty. There was little evidence of tracheal compression by the aneurysm. The patient was discharged on Sept. 3, 1950.

On Sept. 27, 1950, the patient was readmitted complaining of weakness and increasing dysphagia. There were no respiratory symptoms. Bronchoscopy was performed on Oct. 2, 1950. This demonstrated some bulging inward of the left tracheal wall, but an adequate airway was present. During the evening of Oct. 2, 1950, the patient began to vomit bright blood and to pass tarry stools. Bleeding continued, and he died on Oct. 4, 1950. We assumed that the aneurysm had ruptured into the esophagus. Autopsy was not permitted.

DISCUSSION

The satisfactory maintenance of a posterior mediastinal tracheotomy for more than four months in a patient who had profuse, purulent, pulmonary secretions complicating severe tracheal obstruction has demonstrated the feasibility of respiration by this route. Obviously, careful nursing care was of the utmost importance in the result obtained. The right-angled catheter that was necessarily used for this patient was not an ideal tracheotomy tube for the purpose. Had it been possible to employ a tube with a more gradual curve, aspiration of secretions from the bronchial tree would have been facilitated. Despite the angulation of the catheter, however, the tube remained unobstructed except for one episode early in the patient's postoperative course when the nursing technique for the care of the tube was being evolved. Every effort was made to prevent secretions from drying in the tube. During the time when oxygen was necessary, it was administered with "cold steam." In addition, at one-, two-, or three-hour intervals the tracheotomy tube was irrigated with 2 c.c. of warm isotonic saline containing 80,000 units of penicillin. Local instillation of this solution, combined with the systemic use of penicillin and streptomycin, aided in progressively diminishing the quantity and purulent character of the bronchial secretions.

Although the tracheotomy tube had been in place more than four months, a few hours after it had been removed air could not be blown out of the sinus track and during subsequent bronchoscopic examinations it became difficult to visualize the tracheotomy site. In all probability the freeing of the embedded trachea from its site in the wall of the aneurysm contributed more to the restoration of an adequate tracheal lumen than the fibrosis following wrapping of the aneurysm with polythene film.

Mediastinal decompression carried out early in the patient's hospital stay might have been equally effective in relieving his tracheal obstruction. Certainly any attempt to produce a lung fistula or bronchial fistula would have been a much less satisfactory solution of this desperate problem.

SUMMARY

Posterior mediastinal tracheotomy was carried out in a patient with low tracheal obstruction from an aneurysm of the arch of the aorta. Respiration was maintained satisfactorily by this means for more than four months. During this period left and right thoracic explorations were performed in order to wrap the aneurysm with polythene film. The tracheotomy tube was removed and the patient breathed normally for one month. Death then occurred from hemorrhage following erosion of the aneurysm into the esophagus.

REFERENCES

1. Yeager, George H., and Cowley, R. Adams: Studies on the Use of Polythene as a Fibrous Tissue Stimulant, Ann. Surg. **128:** 509-520, 1948.
2. Abbott, Osler A.: Clinical Experiences with the Application of Polythene Cellophane upon Aneurysms of the Thoracic Vessels, J. THORACIC SURG. **18:** 435-461, 1949.
3. Borrie, John, and Griffin, Selwyn G.: Twenty-Seven Cases of Syphilitic Aneurysm of the Thoracic Aorta and Its Branches, Thorax **5:** 293-324, 1950.
4. Gluck, Th.: Die Entwickelung der Lungenchirurgie, Arch. f. klin. Chir. **83:** 581-601, 1907.
5. Eloesser, Leo: Bronchial Stenosis, J. THORACIC SURG. **1:** 194-213, 270-295, 373-396, 485-501, 1931-32.
6. Sauerbruch, Ferdinand: Die Chirurgie der Brustorgane, ed. 2, Berlin, 1920, Julius Springer, vol. 1, p. 851.
7. Rethi, Aurel: Ueber Bronchotomie bei tiefen unheilboren Verengerungen der Luftrohre, München. med. Wchnschr. **69:** 81-83, 1922.
8. Minkin, S.: Experimentelle Begrundung der Bronchostomia transpleuralis, Deutsche Ztschr. f. Chir. **193:** 286-294, 1925.
9. Majewski, Adam: Lung and Bronchial Fistulae as Substitute Respiratory Passages, Przegl. lek. Nr. 9, 151-153, 1912.
10. Kuttner, Hermann: Druckdifferenz—Operationen, Beitr. z. klin. Chir. **60:** 1-93, 1908.
11. Jirasek, Arnold: Posterior Opening of Trachea and Bronchi as Operation of Necessity, Časop. lék. česk. **69:** 1201-1204, 1930.
12. Guleke, N.: Ueber die entlastende Mediastinotomie beim Aneurysma des Aortenbogens, Beitr. z. klin. Chir. **125:** 270-276, 1922.

A BULLET EMBOLUS TO THE LEFT FEMORAL ARTERY FOLLOWING A THORACIC GUNSHOT WOUND

Probable Entrance Through Thoracic Aorta

Case Report and Résumé of Peripheral Arterial Bullet Emboli

John L. Keeley, M.D.
Chicago, Ill.

THE popular and reasonable concept that a bullet wound of the heart or of the great vessels connected with it is rapidly fatal due to hemorrhage has a number of exceptions. The literature contains many interesting and intriguing reports concerning the mode of entrance and behavior of intracardiovascular foreign bodies. Those in which there was peripheral arterial embolization of the foreign body are summarized and such a case is herewith reported.

CASE REPORT

O. H., a Negro laborer aged 27 years, entered Cook County Hospital as an emergency case at 5 A.M. on Nov. 13, 1944, complaining of pain in his left groin and leg and inability to walk. While in a standing position half an hour earlier he had been shot twice, in the right arm and right flank, had immediately noticed pain in his left groin and soon thereafter pain throughout his entire left leg. He was able to take two or three steps before his left leg buckled under him and he fell to the floor.

On admission to the hospital, he was in fairly good general condition. His temperature was 99° F., pulse rate measured 96 beats per minute, respiratory rate 32 per minute. The blood pressure was 124/80. One bullet entered just anterior to the right greater trochanter. The other bullet coursed through the soft tissues posterior to the right humerus at its midpoint and entered the chest through the fifth interspace in the posterior axillary line. There was no Horner's syndrome. The pupils were round, equal, and reacted to light and accommodation. The heart was entirely normal. There was dullness at the base of the right lung posteriorly with diminished breath sounds. The abdomen was soft, and peristaltic sounds were present. The right leg was negative. The left leg was cold and paralyzed below the level of the knee. There was a pulsation in the left femoral artery just below the Poupart's ligament, but none in the popliteal artery nor in the posterior tibial or dorsalis pedis arteries.

Erythrocytes numbered 4,100,000, and the hemoglobin level measured 75 per cent normal. The Kahn test was later found to be negative. Roentgenograms showed no foreign body in the chest where one was expected, but the shadows of two bullets were present in the film of the pelvic region. The bullet

From the Department of Surgery, Stritch School of Medicine, Loyola University, and the Surgical Service of Cook County Hospital.
Received for publication March 5, 1951.

which entered the right hip lay along the inferior ramus of the right pubic bone. The other bullet lay in the position occupied by the left femoral artery immediately over the head of the left femur at the point where the profunda artery arises (Fig. 1).

The nature of the disturbance in the left leg was not immediately realized, and it was thought that the bullet which entered the region of the right hip had damaged the lumbosacral plexus on the left, resulting in paralysis. A neurosurgical consultant felt that the findings could be accounted for entirely on the basis of vascular occlusion. Although approximately eighteen hours had elapsed since the accident, it was considered advisable to remove the bullet.

Fig. 1.—Roentgenogram showing positions of two bullets. The foreign body shadow overlying the head of the left femur represents a bullet in the femoral artery.

Under satisfactory local anesthesia the left femoral artery was exposed. The bullet was found in the artery just at the point where the profunda branch arises (Fig. 2). The bullet was extracted and a tail clot was removed. The artery was closed with a continuous mattress suture of silk everting the edges of the arteriotomy wound. The patient's general condition was satisfactory after the operation. Postoperatively no anticoagulant therapy was immediately available and none was used in the subsequent management. The patient was treated for a time by continuous caudal anesthesia, Novocain injections of the lumbar sympathetic trunk, together with papaverine and the use of venous occlusion in order to dilate the collateral circulation.

Approximately ten days after removal of the bullet, gangrene of the toes developed and gradually extended to involve the leg below the knee. Before a line of demarcation appeared considerable infection in the gangrenous leg occurred. The temperature ranged from 103° F. to 105° F., and the blood culture on one occasion was positive for staphylococcus aureus. Twenty-two days after the accident a supracondular amputation was performed. The wound healed by primary intention and the patient was dismissed from the hospital on the fortieth day.

Sixteen months after the accident the patient was in good general condition and was using an artificial leg satisfactorily. The blood pressure readings in both arms were normal and equal as were the pulses. Fluoroscopic examination of the chest revealed no dilatation of the aorta, no enlargement of the mediastinal shadows, and a clear retrocardiac space. A roentgenogram of the pelvis showed the bullet in place along the inferior ramus of the right pubic bone. Recent efforts to trace this patient have been unsuccessful.

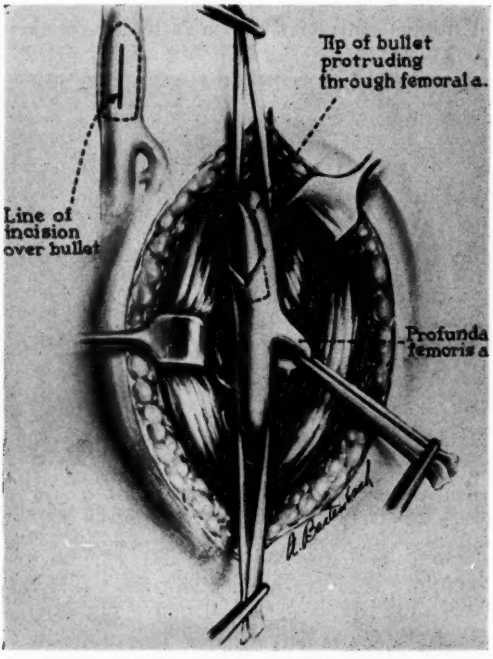


Fig. 2.—Sketch of the operative field showing position of bullet at time of embolectomy.

ABSTRACTS OF PREVIOUSLY REPORTED CASES

Twenty-two cases gleaned from the available literature are summarized. Additional details of the cases presented by Straus[1] appear to justify their inclusion in this report.

The following six patients survived bullet wounds of the chest with peripheral arterial embolization of the missile:

CASE 1.—Schloffer,[2] 1903. A coachman, aged 18 years, shot himself in a suicide attempt while driving a carriage. He was admitted to the hospital two hours later, unconscious and in shock, with rapid and shallow respirations. The heart sounds were normal; a faint pulse was felt at the left wrist but none on the right. The wound of entrance was just to the left of the midline of the sternum at the level of the nipples. No wound of exit

was found. The next morning the patient was conscious and complained of severe pain in his right arm, which was cyanotic, cooler than the left, and had no pulse. Motor and sensory paralysis was present below the level of the elbow. A foreign body, the bullet, was palpated high in the right axilla. Two days later there was fever. Dullness was present at the base of the right lung for about ten days. On the twenty-fourth day roentgenograms showed the bullet to be in the right axillary region.

At operation two months after injury a 7 millimeter bullet with an attached thrombus and "a few drops of pus" were found in the axillary artery at its beginning. The segment of the artery containing the bullet was removed and the ends ligated. There was some improvement in the arm but disturbed motility persisted. There was enough disturbance in sensation so that the patient unwittingly burned his fingers. Two years later he was examined by Prof. von Jaksch, the internist, who found the heart to be enlarged, and findings diagnostic of mitral stenosis and insufficiency were present. The hand and arm were unchanged.

CASE 2.—Rubesch,[3] 1912. A man, aged 28 years attempted suicide. The bullet entered between the third and fourth interspace 1.5 centimeters medial to the left mammary line. He remained standing, developed dyspnea, and when seen in the clinic one hour later had severe pain in the entire right leg, which was cooler than the left. Both femoral pulses were present. Roentgenograms revealed no bullet in the chest, but the shadow of the bullet was found just inferior to the head of the right femur, 5 to 6 cm. below the inguinal ligament. Embolectomy two days after the injury revealed a thrombus proximal and distal to the bullet, which lay transverse in the femoral artery. Discoloration of the foot began two days after embolectomy. Three amputations were performed; the first, fifteen days after the shooting, the last, above the knee, two months later. Two and one-half months after discharge from the hospital an x-ray of the chest was normal, but the patient had a systolic murmur at the apex of the heart. The bullet was thought to have gained entrance to the circulation by way of the heart.

CASE 3.—Boeckel,[4] 1917. An infantryman was shot in the right shoulder at the level of the deltoid insertion, and subsequently developed hemoptysis. On the third day roentgenograms showed the missile to be located in the left ventricle. The bullet embolized to the left femoral artery on the fourteenth day, at which time embolectomy was done. Gangrene of the leg developed, and it was amputated on the eighteenth day. The patient was dismissed from medical care on the thirty-fourth day. The missile was described as "a German bullet."

The case of Maurizot[5] so closely resembles that of Boeckel as to the missile, "a German bullet," the point of entrance, symptoms, the intervals before embolectomy, gangrene, amputation, and dismissal that the two authors seem to have described the same case independently.

CASE 4.—Perdoux' case, presented by Baumgartner,[6] 1919. A soldier, age not stated, was wounded by an airplane machine gun bullet which entered in the right posterior axillary line just lateral to the scapula. He was unconscious for a time but was in good general condition when examined nine days later. On the thirteenth day, roentgenograms showed the missile to be in the anterior part of the heart near the diaphragm. The point of the bullet was directed toward the base of the heart. Twenty days after injury roentgenograms and fluoroscopy showed the bullet in the same region with the point immobile, but the base oscillated with the cardiac movements. Thirty days after injury the missile was absent from the cardiac area, but was seen in the region of the right common iliac artery. The migration had not caused any symptoms. There were no pulses palpable in the right leg, and the blood pressure at the level of the calf was 50/30. On the forty-third day 1,200 cubic centimeters of serosanguineous fluid were removed from the right pleural cavity. Culture of it was negative and the temperature was normal. On the eighty-seventh day roentgenograms

showed the bullet lying obliquely in the region of the right common iliac artery. At that time the patient had no dyspnea and walked easily but had cramps in his leg while climbing stairs. On Dec. 21, 1918, ninety days after the accident, laparotomy was performed, the right iliac artery was opened, and the bullet was removed. The artery was sutured, but the circulation was not restored. Pulsations did not return but the efficiency of the circulation improved. Six months after injury the patient could walk fast without discomfort, but noted weakness in the leg on climbing stairs. There were no cardiac symptoms and no abnormal cardiac findings.

The author thought that the early immobility of the bullet meant that the point was fixed in the anterior wall of the ventricle and the base in the posterior wall. The movements of the heart loosened the base and as it gradually entered the cavity of the vertical the wound in the posterior wall healed behind it. The base was then free to move in the cavity of the left ventricle. It was not until the movements of the heart freed the point of the bullet from the anterior wall of the ventricle that embolization occurred.

CASE 5.—LaRoque,[7] 1926. A male, aged 36 years, was shot from above while running downstairs. He continued to run "a short distance" when the left leg gave way; pain and weakness developed in it. Examination disclosed the bullet hole medial to the ventricle border of the left scapula, and a large effusion in the left thorax. Roentgenograms showed bullet fragments and fluid in the left chest, but the main part of the bullet was found by roentgenogram in the region of the left femoral artery. The patient was treated symptomatically and, on the eightieth day after the accident, 2 inches of the left femoral artery containing the bullet and a similar length of the accompanying vein were resected just above the profunda branch. Quadruple ligation of the vessels was done. The postoperative course was uneventful, and the patient was ambulatory in two weeks. Efforts to trace this patient after dismissal were unsuccessful.

CASE 6.—Cooper and co-workers,[8] 1948. A soldier, aged 22 years, sustained a penetrating wound of the left arm and chest from a 45 caliber bullet on May 26, 1945. He was admitted to a hospital shortly thereafter. The bullet penetrated the lateral aspect of the left arm, fractured the humerus, emerged on the medial aspect near the axilla, and then entered the chest between the fourth and fifth ribs in the axillary line. A hemothorax developed. The fracture of the humerus was treated and the wound of the chest was débrided and sutured. A swishing murmur was heard over the sternum on the fifteenth day. On the twenty-sixth day he suddenly complained of pain, coldness, and numbness in the right foot. The femoral pulse on the right disappeared the next day, and the pulsations in the left leg were diminished. Roentgenograms three days later showed the bullet at the bifurcation of the aorta. Seven months later, after two attacks of pain in the chest and abdomen, the segment of the abdominal aorta containing the bullet just above the bifurcation was excised. Complete recovery followed. The bullet apparently entered the heart or was carried there from one of the great vessels, and remained there for twenty-six days.

The following are ten fatal cases of thoracic bullet wounds with peripheral arterial embolization of the missile.

CASE 7.—Schmidt,[9] 1885. A man, aged 30 years, committed suicide. The body was studied in an anatomic laboratory, and no details of the clinical course are available. The bullet was 3 cm. long, 2 cm. in circumference and weighed 3.45 Gm. It entered the sixth interspace 10 centimeters to the left of the sternum, passed upward and through the anterior wall of the left pulmonary vein, and caused slight damage to the posterior wall. It was then carried by the blood stream into the left auricle through the left ventricle and became lodged in the left femoral artery, where it was associated with a thrombus 15 cm. long, indicating a period of survival. The cause of death was thought to be pulmonary hemorrhage, and the author states that he had not found a similar case previously reported.

CASE 8.—Dittrich,[10] 1897. The author reports only the following details which were apparently found at autopsy following suicide. There was a conical bullet low in the abdominal aorta. It had penetrated the anterior wall of the ascending aorta, reached the aortic lumen, and probably descended only on the manipulation of the cadaver during transportation.

CASE 9.—O'Neill,[11] 1917. A soldier sustained a wound from a shell fragment in the left posterior axillary line at the level of the fifth interspace. Roentgenograms three days later showed paralysis of the left half of the diaphragm, a left pleural effusion, and a foreign body probably in the left ventricle. A half hour after the roentgenograms the left leg became cold and cyanotic from the knee to the ankle. Roentgenograms on the fourth day showed the foreign body to be absent from its previous thoracic location, but present in the midabdomen apparently at the aortic bifurcation. The patient died on the fifth day of "generalized gas gangrene," and at autopsy the bullet was found at the bifurcation of the left common iliac artery. The pericardium had a small tear with a small amount of blood in it. A tear of the interventricular septum and of the cordae tendinae of the aortic valve indicated probable entrance into the left ventricle.

CASE 10.—De Rouault,[12] 1917. A soldier sustained a bullet wound, caliber not stated, at the external end of the spine of the right scapula. There was shock, hemoptysis, a right hemothorax, and motor and sensory paralysis of the left leg. A roentgenogram showed no foreign body in the spinal, thoracic, or abdominal regions, but a roentgenogram on the fourth day showed the bullet in the region of the left femoral artery. The treatment was supportive, and the bullet was removed on the fourth day from the left femoral artery under local anesthesia. The patient died on the sixth day and autopsy showed a right hemothorax, congestion of the base of the left lung, a transverse wound of the lower lobe of the right lung, and gangrene of the left leg. The aortic wound was an irregular triangular defect at the junction of the arch and the descending aorta. Its greatest dimension was 5 millimeters on the outside and 15 millimeters on the inside. A small superficial wound of the intima was present on the opposite wall. There was a slight periaortic effusion of blood.

CASE 11.—Bland-Sutton,[13] 1919. A soldier was struck in the "chest" by a rifle bullet, caliber not stated. The bullet was palpated in the left external iliac artery before death. A thoracentesis was done. Four pints of "fused blood" were recovered. The bullet wound was found in the left external iliac artery at autopsy, and there was a "small wound in the aorta just below the arch." Death was thought to be due to streptococcal infection as a positive culture was obtained.

CASE 12.—LeClerq and Muller,[14] 1930. A man, after being wounded by an 8 millimeter bullet, 3 centimeters to the left of the midline of the back, at the level of the eighth dorsal vertebra, brought up "frothy blood" and died after walking "a few steps." Autopsy showed the bullet had passed through the muscles, grooved the left surface of the seventh dorsal vertebrae, passed through the thoracic aorta, pierced the left bronchus but could not be found anywhere in the thoracic viscera. There was hemorrhage into the posterior mediastinum down to the diaphragm. The bullet was found in the left femoral artery where the profunda arises. Apparently the bullet fell back into the aorta after causing an opening in the left bronchus.

CASE 13.—LeClerq and Muller,[14] 1930. A woman, aged 27 years, sustained a wound from a "ball," size not stated, fired from a distance of about 6 meters. It pierced the upper part of the left thorax near the axillary line. The survival period was not stated, but autopsy the "next day" disclosed the bullet had pierced the left lung causing abundant hemorrhage into the pleural cavity, pericardium, and mediastinum. There was erosion of

part of the pericardium, but there was no trace of the bullet in the thoracic viscera. It was found in the left femoral artery at the beginning of Hunter's canal and at right angles to the axis of the artery where it had caused subendothelial ecchymosis. There was a "small discrete aortic lesion seen with difficulty," apparently in the ascending aorta.

CASE 14.—Walcher,[15] 1932. A man, aged 30 years, was shot at a distance of "several steps" by a 5 millimeter lead bullet in the third intercostal space 1 centimeter to the left of the sternum. Roentgenograms of the chest showed no bullet. He survived nineteen hours and his death was ascribed to cardiac tamponade, but signs and symptoms of peritonitis were also present. Autopsy showed fibro-purulent peritonitis. There was a 4 millimeter opening in the pericardium, and in the anterior wall of the ascending aorta, and injury to the intima of the posterior wall of the aorta at the same level. The bullet was found in the left profunda artery 6 centimeters below its origin.

CASE 15.—Piedelievre and Etienne-Martin,[16] 1933. A young man died suddenly after being shot from a distance of one to two meters with an 8 millimeter bullet. It perforated the sternum at the level of the third intercostal space. Autopsy disclosed that the bullet had passed through the pericardium and the anterolateral wall of the aortic arch. The heart and lungs were intact, the periaortic and pericardiac hemotomas were extensive. The trachea and esophagus were unharmed, and the pleural cavities contained no blood. The cause of death was hemorrhage. The bullet was found in the left femoral artery at the origin of the profunda branch.

CASE 16.—Gonzales,[17] 1937. A man, aged 28 years, was shot with a 38 caliber lead bullet while fleeing the police. The missile entered the back two inches to the right of the midline at the level of the seventh dorsal vertebra. The patient experienced severe pain, and motor and sensory paralysis below the level of the seventh dorsal segment came on suddenly. He was given supportive treatment and survived thirty-two hours. Autopsy disclosed that the bullet had traveled a distance of three inches from the skin to the posterior wall of the aorta passing through the body of the seventh dorsal vertebra and severing the spinal cord at this level. There was extensive periaortic and intrapleural hemorrhage, the right and left pleural cavities containing 600 and 2,800 cubic centimeters of fluid and clotted blood, respectively. The wound in the aorta was not described. The bullet was found in the left common iliac artery two inches below the bifurcation of the aorta. The cause of death was given as hemorrhage, hemothorax, and pulmonary collapse.

The following five cases describe bullet wounds in which the damage was principally abdominal, the missile entering the abdominal aorta and embolizing from there.

CASE 17.—Jecks,[18] 1890. A woman, aged 36 years, committed suicide and expired on admission. The first bullet, weighing 148 grains, entered just below the xyphoid cartilage, pierced the falciform ligament, the left lobe of the liver, and came to rest above the posterior part of the left iliac crest causing ecchymosis at that point. The second bullet entered the lower part of the left chest by passing through the sixth intercostal space at the level of the xyphoid cartilage. It grazed the pericardium and a portion of the left lung, passed through the diaphragm and the left lobe of the liver, made a furrow in the superior border of the pancreas, and penetrated the anterior wall of the aorta just above the celiac axis leaving an opening which admitted a fingertip. The bullet which weighed 169 grains was found at the bifurcation of the aorta. The period of survival was estimated to be one and one-half hours. The abdominal cavity was filled with blood.

CASE 18.—Lambotte and Herman,[19] 1895. A man, aged 19 years, sustained a self-inflicted bullet wound in his epigastrium. He fainted, but was conscious when admitted to a hospital three hours later. He was pale, but the pulse was of good quality and regular. He complained of abdominal pain and vomited large quantities of bile. The wound of entrance was in the epigastrium to the right of the midline. Three and one-half hours after the accident laparotomy disclosed a through-and-through wound of the liver, and perforation of the stomach walls in the prepyloric region. The bullet was not found. Death occurred five hours later, and at the autopsy the anterior wall of the aorta was found to have been perforated by the bullet just below the celiac axis. After an hour's search in this area the 9 mm. bullet was found in the left common iliac artery just proximal to the origin of the left internal iliac artery. The bullet was not deformed.

CASE 19.—Kratter,[20] 1906. A young woman attempted suicide by inflicting a bullet wound in the lower abdomen. Bluish discoloration, pain, and tingling developed in the left foot. At laparotomy the next day no injury of any abdominal organ was noted and the bullet was not found. The dorsalis pedis pulse was absent, and the femoral pulse was weak on the left. Gangrene developed and an amputation of the left leg was done at the knee. The gangrene spread to midthigh. Cystitis and pyelonephritis ensued, and the patient died on the fifty-seventh day in uremia. At autopsy the bullet which had entered the abdominal aorta just above the bifurcation was found in the left common iliac artery which it occluded.

CASE 20.—Momm,[21] 1910. A man, aged 23 years, was shot at a distance of three meters; the bullet entered just inferior and to the left of the xyphoid cartilage. There was severe shock and signs of internal hemorrhage. During laparotomy a tamponade was placed between the stomach and the liver to control severe hemorrhage, the exact source of which was unidentified. The patient improved after the operation, but on the twelfth day an aneurysm appeared in the left groin, and this was followed by progressive gangrene of the left leg. At operation on the nineteenth day the bullet was found lying between the femoral artery and vein in a false aneurysmal sac. The left leg became gangrenous to a point above the knee, and the patient died of sepsis on the twenty-seventh day. Autopsy disclosed the wound of entrance in the anterior surface of the aorta to be 7 by 3 millimeters, a 6 millimeter slit in the intima and media of the posterior wall of the aorta was present. There was a bullet wound of the liver with encapsulation of bile in the area. A firm clot was found in the left iliac artery 5 centimeters below the bifurcation of the aorta. A false aneurysm of the medial side of the left femoral artery due to erosion by the bullet was found together with thrombosis of the veins of the left leg, gangrene extending above the left knee, and abscesses in the liver, kidneys, spleen, and pericardium.

CASE 21.—Baker,[22] 1935. A man, aged 23 years, was shot in the right flank in the posterior axillary line between the eleventh and twelfth ribs by a 9 by 16 millimeter bullet. The patient was in shock, and the right leg was painful and limp. He died within four hours. Autopsy revealed 150 cubic centimeters of blood in the peritoneal cavity, a large retroperitoneal hematoma above the right kidney, and a groove across the lower pole of the right kidney. The bullet had severed the right ureter and traversed the psoas muscle. A transverse tear 3 cm. above the bifurcation of the aorta and involving two-thirds of the circumference of the vessel was found. It spared only a portion of the wall on the right side. The bullet lay in the right femoral artery. The cause of death was hemorrhage.

CASE 22.—Oberhelman and LeCount,[23] 1936. A middle-aged man was shot by his son, the caliber of the bullet not reported. It caused a through-and-through wound in the soft part of the left upper arm, and entered the chest at the level of the tenth rib in the posterior axillary line. Severe pain was experienced in the left foot and was soon followed by motor and sensory paralysis of the left leg. An embolectomy was done, and on the

part of the pericardium, but there was no trace of the bullet in the thoracic viscera. It was found in the left femoral artery at the beginning of Hunter's canal and at right angles to the axis of the artery where it had caused subendothelial ecchymosis. There was a "small discrete aortic lesion seen with difficulty," apparently in the ascending aorta.

CASE 14.—Walcher,[15] 1932. A man, aged 30 years, was shot at a distance of "several steps" by a 5 millimeter lead bullet in the third intercostal space 1 centimeter to the left of the sternum. Roentgenograms of the chest showed no bullet. He survived nineteen hours and his death was ascribed to cardiac tamponade, but signs and symptoms of peritonitis were also present. Autopsy showed fibro-purulent peritonitis. There was a 4 millimeter opening in the pericardium, and in the anterior wall of the ascending aorta, and injury to the intima of the posterior wall of the aorta at the same level. The bullet was found in the left profunda artery 6 centimeters below its origin.

CASE 15.—Piedelievre and Etienne-Martin,[16] 1933. A young man died suddenly after being shot from a distance of one to two meters with an 8 millimeter bullet. It perforated the sternum at the level of the third intercostal space. Autopsy disclosed that the bullet had passed through the pericardium and the anterolateral wall of the aortic arch. The heart and lungs were intact, the periaortic and pericardiac hemotomas were extensive. The trachea and esophagus were unharmed, and the pleural cavities contained no blood. The cause of death was hemorrhage. The bullet was found in the left femoral artery at the origin of the profunda branch.

CASE 16.—Gonzales,[17] 1937. A man, aged 28 years, was shot with a 38 caliber lead bullet while fleeing the police. The missile entered the back two inches to the right of the midline at the level of the seventh dorsal vertebra. The patient experienced severe pain, and motor and sensory paralysis below the level of the seventh dorsal segment came on suddenly. He was given supportive treatment and survived thirty-two hours. Autopsy disclosed that the bullet had traveled a distance of three inches from the skin to the posterior wall of the aorta passing through the body of the seventh dorsal vertebra and severing the spinal cord at this level. There was extensive periaortic and intrapleural hemorrhage, the right and left pleural cavities containing 600 and 2,800 cubic centimeters of fluid and clotted blood, respectively. The wound in the aorta was not described. The bullet was found in the left common iliac artery two inches below the bifurcation of the aorta. The cause of death was given as hemorrhage, hemothorax, and pulmonary collapse.

The following five cases describe bullet wounds in which the damage was principally abdominal, the missile entering the abdominal aorta and embolizing from there.

CASE 17.—Jecks,[18] 1890. A woman, aged 36 years, committed suicide and expired on admission. The first bullet, weighing 148 grains, entered just below the xyphoid cartilage, pierced the falciform ligament, the left lobe of the liver, and came to rest above the posterior part of the left iliac crest causing ecchymosis at that point. The second bullet entered the lower part of the left chest by passing through the sixth intercostal space at the level of the xyphoid cartilage. It grazed the pericardium and a portion of the left lung, passed through the diaphragm and the left lobe of the liver, made a furrow in the superior border of the pancreas, and penetrated the anterior wall of the aorta just above the celiac axis leaving an opening which admitted a fingertip. The bullet which weighed 169 grains was found at the bifurcation of the aorta. The period of survival was estimated to be one and one-half hours. The abdominal cavity was filled with blood.

CASE 18.—Lambotte and Herman,[19] 1895. A man, aged 19 years, sustained a self-inflicted bullet wound in his epigastrium. He fainted, but was conscious when admitted to a hospital three hours later. He was pale, but the pulse was of good quality and regular. He complained of abdominal pain and vomited large quantities of bile. The wound of entrance was in the epigastrium to the right of the midline. Three and one-half hours after the accident laparotomy disclosed a through-and-through wound of the liver, and perforation of the stomach walls in the prepyloric region. The bullet was not found. Death occurred five hours later, and at the autopsy the anterior wall of the aorta was found to have been perforated by the bullet just below the celiac axis. After an hour's search in this area the 9 mm. bullet was found in the left common iliac artery just proximal to the origin of the left internal iliac artery. The bullet was not deformed.

CASE 19.—Kratter,[20] 1906. A young woman attempted suicide by inflicting a bullet wound in the lower abdomen. Bluish discoloration, pain, and tingling developed in the left foot. At laparotomy the next day no injury of any abdominal organ was noted and the bullet was not found. The dorsalis pedis pulse was absent, and the femoral pulse was weak on the left. Gangrene developed and an amputation of the left leg was done at the knee. The gangrene spread to midthigh. Cystitis and pyelonephritis ensued, and the patient died on the fifty-seventh day in uremia. At autopsy the bullet which had entered the abdominal aorta just above the bifurcation was found in the left common iliac artery which it occluded.

CASE 20.—Momm,[21] 1910. A man, aged 23 years, was shot at a distance of three meters; the bullet entered just inferior and to the left of the xyphoid cartilage. There was severe shock and signs of internal hemorrhage. During laparotomy a tamponade was placed between the stomach and the liver to control severe hemorrhage, the exact source of which was unidentified. The patient improved after the operation, but on the twelfth day an aneurysm appeared in the left groin, and this was followed by progressive gangrene of the left leg. At operation on the nineteenth day the bullet was found lying between the femoral artery and vein in a false aneurysmal sac. The left leg became gangrenous to a point above the knee, and the patient died of sepsis on the twenty-seventh day. Autopsy disclosed the wound of entrance in the anterior surface of the aorta to be 7 by 3 millimeters, a 6 millimeter slit in the intima and media of the posterior wall of the aorta was present. There was a bullet wound of the liver with encapsulation of bile in the area. A firm clot was found in the left iliac artery 5 centimeters below the bifurcation of the aorta. A false aneurysm of the medial side of the left femoral artery due to erosion by the bullet was found together with thrombosis of the veins of the left leg, gangrene extending above the left knee, and abscesses in the liver, kidneys, spleen, and pericardium.

CASE 21.—Baker,[22] 1935. A man, aged 23 years, was shot in the right flank in the posterior axillary line between the eleventh and twelfth ribs by a 9 by 16 millimeter bullet. The patient was in shock, and the right leg was painful and limp. He died within four hours. Autopsy revealed 150 cubic centimeters of blood in the peritoneal cavity, a large retroperitoneal hematoma above the right kidney, and a groove across the lower pole of the right kidney. The bullet had severed the right ureter and traversed the psoas muscle. A transverse tear 3 cm. above the bifurcation of the aorta and involving two-thirds of the circumference of the vessel was found. It spared only a portion of the wall on the right side. The bullet lay in the right femoral artery. The cause of death was hemorrhage.

CASE 22.—Oberhelman and LeCount,[23] 1936. A middle-aged man was shot by his son, the caliber of the bullet not reported. It caused a through-and-through wound in the soft part of the left upper arm, and entered the chest at the level of the tenth rib in the posterior axillary line. Severe pain was experienced in the left foot and was soon followed by motor and sensory paralysis of the left leg. An embolectomy was done, and on the

fourth day an amputation because of gangrene of the left leg was performed. The patient died soon thereafter. Autopsy showed a bullet wound of entrance 8 millimeters in diameter 4 millimeters cephaled to the origin of the left renal artery. There was a longitudinal tear in the intima of the aorta 12 millimeters long just to the right of the origin of the celiac axis. There were 732 Gm. of blood in the left pleural space and an extensive retroperitoneal hematoma. The causes of death were hemorrhage, shock, and sepsis.

DISCUSSION

Of the twenty-three cases in this report, one was in the second decade, seven were in their third decade, four were in their fourth decade, and the age was not stated in the remainder. Two were women. Information concerning the size of the missile was available in only nine of the cases, and in these it was small. The wound of entrance was in the anterior chest wall in nine cases, in the posterior chest wall in five. The bullet passed through the upper arm before entering the chest in two cases and the flank in a third. Seven of the bullets entered the chest to the left of the sternum and two passed through it. Three of the five posterior chest wounds were to the left of the midline, the others were on the right.

The chief symptoms and findings in those patients who survived a bullet wound of the heart or its associated great vessels were shock and hemorrhage. The more or less prompt embolization of the missile in some of the cases resulted in early signs and symptoms of motor and sensory paralysis due to the ischemia. The left leg was thus involved in the cases of De Rouault,[12] LaRougue,[7] Oberhelman and LeCount,[23] and in my case. The right leg was involved in the cases of Rubesch[3] and Baker,[22] and the right arm in Schloffer's[2] case. The effects of the ischemia prompted the tentative diagnosis of spinal cord injury in the case of De Rouault and in my case.

The significant laboratory procedures reported in these cases have been the x-ray studies. In the cases of De Rouault,[12] Rubesch,[3] Walcher,[15] and the case reported here roentgenograms of the chest failed to reveal the presence of the suspected foreign body. In LaRoque's[7] case fragments of the missile remained in the region of the wound of entrance, but the main part was shown to be in the region of the left groin. The shadow of the missile in the case of Rubesch and in the one reported herein overlay the region of the head of the femur, and therefore was in the region of the corresponding femoral artery. X-ray studies in Schloffer's case (1903) disclosed the bullet to be in the region of the right axillary artery.

The missile embolized to the right iliac or femoral artery in two cases, to the left iliac or femoral artery in fourteen cases, and was found near or at the bifurcation of the aorta in three cases. In Schloffer's case it was carried to the right axillary artery. The predominance of cases in which the missile embolized to the left iliac or femoral artery is in keeping with the studies of LeClerq and Muller.[14] They found that the angles formed by the iliac vessels as they arise from the aorta average 65 degrees in the male and 75 degrees in the female. Their studies further showed that the right iliac vessels form an angle with the midline of about 45 degrees, whereas the angle formed by the midline and the

left iliac vessels averages 30 degrees. The left iliac artery therefore more nearly approximates a direct continuation of the line of the aorta. Whether the missile remains in the aorta, the iliac or the femoral artery depends on its size.

The treatment has been confined to three phases, the first of which is supportive to combat the effects of hemorrhage and shock. The prompt and generous use of blood and plasma has made this more effective. The second phase consists of direct attack on the foreign body. Embolectomy was done in Boeckel's[4] case fourteen days after injury but the first day after embolization. Embolectomy was done two days after embolization in Rubesch's case, in the case of Oberhelman and LeCount[23] after one day, and in my case after twenty hours. LaRoque's patient was treated by resection of the segment of the left femoral artery obstructed by and containing the missile together with a corresponding segment of femoral vein followed by quadruple ligation. This was done on the eightieth day after injury, and the collateral circulation was entirely adequate. About six months after embolization, the patient reported by Cooper et al[8] underwent resection of the segment of the aorta containing the foreign body just above the bifurcation. Schloffer's case was treated by resection also, the segment of the right axillary artery containing the bullet being excised.

The third phase of treatment has consisted of amputation for gangrene. Rubesch's case had three amputations before healing was satisfactory. The patient survived. In Boeckel's surviving case amputation was done on the eighteenth day, four days after embolectomy. The patient described by Oberhelman and LeCount[23] had an amputation four days after injury and died soon thereafter. The patient described herein had a supracondylar amputation on the twenty-second day after injury and recovered.

There were fifteen fatal cases; nine were due to hemorrhage and shock. Infection appeared to be the cause of death in four and cardiac tamponade in one. The cause was not mentioned in Dittrich's[10] case.

The aortic wounds are of considerable interest. They consist of a small wound in one wall of the aorta with damage of various degrees to the intima and media of the opposite wall, the force of the bullet having been largely spent after passing through one wall. Through-and-through wounds of the aorta (called "binocular" wounds in many of the articles) are compatible with survival for several days, death finally occurring from other causes, as in the cases of Kornblith[24] and Vance.[25] In the case reported by Leclerq and Muller, the bullet, after making two wounds in the aorta, pierced the wall of the left bronchus before falling back into the aorta and embolizing to the left femoral artery.

The point of entrance into the cardiovascular system found at autopsy was the ascending aorta in four cases, the thoracic aorta in three, the aortic arch in one, the abdominal aorta in five, the left pulmonary vein in one, and the posterior wall of the left ventricle in one. Naturally the wounds of the ascending aorta and the aortic arch were associated with the anterior chest wounds; the wounds of the thoracic aorta and the posterior part of the arch were associated with the posterior wounds.

The very interesting experimental work of Fritz and co-workers[26] indicates exceedingly well what may be expected when foreign bodies gain entrance directly or indirectly to the cavities of the heart. Fritz and his associates introduced foreign bodies into the left side of the heart through the left auricular appendage in thirteen dogs. In twelve animals the foreign body was immediately embolized to the aorta or one of its branches. In one animal the foreign body was trapped in the auricle by the ligature used to close it. Of fifteen foreign bodies introduced into the right ventricle thirteen were promptly carried into the pulmonary circuit; one was caught behind the papillary muscles of the right ventricle. No cardiac dysfunction resulted. Autopsy in this animal ten days later showed the foreign body to be encapsulsated by a thin layer of fibrin with no associated thrombus formation. In the other animal, the foreign body remained in the tip of the right auricle and was found to be firmly fixed there without a propagating thrombus at autopsy twelve weeks later.

Foreign bodies were introduced into the jugular vein in seven dogs and in all embolized to the lungs. In a few of the animals the foreign body remained in the chambers of the heart for as long as thirty-six hours.

One would expect a foreign body entering the aorta or left heart chambers to be propelled quickly in the direction of blood flow. However, this did not occur in the case reported by Tegeler.[27] The patient recovered and had no cardic symptoms for thirteen months when he was accidentally electrocuted. Autopsy disclosed that the bullet had entered the aorta, fallen back into the left ventricle where it became encapsulated by fibrin, and was held in place by the chorda tendineae. In Singleton's[28] case a farm laborer was shot accidentally in the anterior part of the left chest. A "state of collapse" followed but the patient returned to work in four days. He died suddenly on the fifty-fourth day after the injury. At autopsy the bullet was found encapsulated in the left ventricle by a thin coat of fibrin, having passed through the right ventricle, the upper portion of the septum, the semilunar valve, and the aortic wall before entering the left ventricle.

SUMMARY

A case of thoracic bullet wound followed by embolization to the left femoral artery is presented. This appears to be the sixth surviving case of its kind, five others having been found in the literature. In addition fifteen other cases in which bullet wounds of the heart or associated great vessels had been followed by peripheral arterial embolization of the foreign body are summarized.

It is obvious from the behavior of the missile both in the clinical cases and in animal experiments that any conclusion as to the point of entrance into the vascular system on the basis of time of embolization is unsound. The entrance of a missile into the cardiovascular system through the wall of a heart chamber or aorta might be determined with greater accuracy on the basis of the presence or absence of hemoptysis, pericardial friction rub, or effusion, murmurs, electrocardiographic changes, and the results of fluoroscopic and x-ray studies.

Detailed study of these cases suggests that a thoracic wound with a small caliber missile may do no serious damage to the lungs and pulmonary vessels

but still may have sufficient force to pierce one wall of the aorta or of a cardiac chamber. It may then be embolized immediately or later to the systemic vessels, being carried usually to the iliac or femoral vessels and more commonly to the left side. The path of a bullet which appears to afford the best chance of entering the aorta without damage to associated structures is apparently through the anterior chest wall to the left of the sternum. A wound involving the abdominal aorta is likewise compatible with survival for a time, but is usually associated with extensive damage to other structures leading to higher mortality, there being no reported cases of survival from this combination of injuries.

Therefore, in instances of bullet wounds with only a wound of entrance, failure to demonstrate the foreign body in the expected region should prompt the suspicion that it may have entered the cardiovascular system and been carried away, thus suggesting that other parts of the body be studied roentgenographically. Early motor and sensory paralysis due to ischemia may suggest the erroneous diagnosis of a lesion of the nervous system. Finally, the correct diagnosis should lead to early embolectomy, a recommendation made by Schloffer in 1903. With the present improvements in the technique of embolectomy, recoveries from aortic or cardiac wounds with embolization of the foreign body might, in the future, be complete even to the survival of the involved extremity.

REFERENCES

1. Straus, R.: Pulmonary Embolism Caused by a Lead Bullet Following a Gunshot Wound of the Abdomen, Arch. Path. **33:** 63, 1942.
2. Schloffer, H.: Bullet Embolism and Remarks Concerning Bullet Injuries of the Heart and Great Vessels, Beitr. z. klin. Chir. **37:** 698, 1903.
3. Rubesch, R.: Embolic Displacement of Projectiles, Beitr. z. klin. Chir. **80:** 394, 1912.
4. Boeckel, M.: Migration of a German Bullet in a Wounded Man, from the Left Ventricle into the Left Femoral Artery, Lyon méd. **126:** 567, 1917.
5. Maurizot, M.: Migration of a Bullet from the Left Heart to the Left Femoral Artery, Univ. de Lyon Theses Méd. N. 100, 1919-1920.
6. Baumgartner, M. A.: Bullet Wound of the Heart: Migration of the Bullet into the Common Iliac Artery, Extraction, Recovery. Bull. et mém. Soc. d. chirurgiens de Paris, **45:** 632, 1919.
7. LaRoque, G. P.: Penetrating Bullet Wound of the Thoracic Aorta Followed by Lodgment of the Bullet in the Femoral Artery, Ann. Surg., **83:** 827, 1926.
8. Cooper, F. W. Jr., Harris, M. H., and Kahn, J. W.: Ligation and Division of the Abdominal Aorta for Metallic Embolus from the Heart: Postoperative Observations of the Circulation in the Extremities, Ann. Surg. **127:** 1, 1948.
9. Schmidt, G. B.: Eine Tödliche Schussverletzung des Gefabsystems mit Embolie des Geschosses, Zentralbl. f. Chir., **12:** 131, 1885.
10. Dittrich, P.: Lehrbuch der gerichtlichen Medicina fur Studivende und aertze. Wein, A. Leipz. W. Braumiller, 1897, p. 44.
11. O'Neill, C. S.: Fragment of Shell in Arterial Circulation, Brit. M. J. **2:** 719, 1917.
12. De Rouault, M.: Gunshot Wound of the Arch of the Aorta. Migration of the Bullet into the Left Femoral Artery, Bull. et mém. Soc. d. chirurgiens de Paris, **43:** 2264, 1917.
13. Bland-Sutton, Sir John: A Lecture on Missiles as Emboli, Lancet, **196:** 773, 1919.
14. LeClercq and Muller: Migratory Bullets, Ann. de Méd. Leg. **10:** 33, 1930.
15. Walcher, K.: Bullet Emboli; Two Case Histories, Zentralbl. f. Chir. **59:** 1220, 1932.
16. Piedelievre, R. and Etienne-Martin, P.: Migratory Projectiles, Presse méd., **41:** 1681, 1933.
17. Gonzales, T. A.: Personal communication.
18. Jecks, C.: A Case of Bullet Wound of the Abdominal Aorta in Which the Bullet Remained in the Lumen of that Vessel, Lancet **1:** 800, 1890.
19. Lambotte, A., and Herman, F.: Wound of the Abdomen by Firearms. Perforation of the Liver, the Stomach and the Aorta; Migration of the Projectile into the Left Common Iliac Artery, Laparotomy, Death and Autopsy, Ann. Soc. Méd. d'anvers **67:** 15, 1895.

20. Kratter: A Rare Bullet Injury, Vrtljschr. f. gerichtl. Med. **31**: 342, 1906.
21. Momm: Ein Schussdurch leber und Aorta, Tod noch vier wochen infalge sepsis, Deutsche med. Wehnschr. **36**: 2422, 1910.
22. Baker, R. D.: Bullet Embolism, Am. J. Surg. **29**: 282, 1935.
23. Oberhelman, H. A., and LeCount, E. R.: Peace Time Bullet Wounds of the Abdomen, Arch. Surg. **32**: 373, 1936.
24. Kornblith, B. A.: Gunshot Wound through the Abdominal Aorta, Ann. Surg., **113**: 637, 1941.
25. Vance, B. M.: Three Unusual Cases of Bullet Wounds of the Heart Showing Attempts at Healing, Am. J. Med. Science **169**: 872, 1925.
26. Fritz, J. M., Newman, M. M., Jampolis, R. W. and Adams, W. E.: Fate of Cardiac Foreign Bodies, Surgery **25**: 869, 1949.
27. Tegeler: Schussverletzung der Aorta mit eingeheilter Kugel in der Herzklappe, München. med. Wehnschr. **56**: 1740, 1909.
28. Singleton, D., Lancet **1**: 33, 1879.

THE EFFECT OF DECREASING THE AMOUNT OF LUNG TISSUE ON THE RIGHT VENTRICULAR PRESSURES IN ANIMALS*

RALPH F. CARLSON, M.D.,** BERNARD C. CHARBON, M.D.,
HARMIA G. A. CHARBON, M.D., AND W. E. ADAMS, M.D.
CHICAGO, ILL.

STUDIES of the dynamics of the lesser circulation have been carried out by many workers since at least 1850. Most of the studies reported have been acute experiments in which the pulmonary vascular bed was gradually occluded. Right auricular or ventricular, or pulmonary artery pressures along with systemic arterial and venous pressures were recorded. The pressures in the lesser circulation were recorded by means of placing a glass tube directly into a branch of the pulmonary artery (Beutner, 1852),[1] or by passing a glass or silver tube through an external jugular vein into the right heart as was reported by Badoud[2] in 1874.

Lichtheim,[3] doing experiments on dogs, using curare and artificial respiration, in Cohnheim's and Heidenhain's laboratories, reported in 1876 that up to three-fourths of the pulmonary vascular bed could be occluded without changes occurring in the carotid or peripheral venous pressures. The mean pulmonary arterial pressure rose as he obstructed more and more of the pulmonary vascular bed. When more than three-fourths of it was occluded, the systemic pressure began to fall, although the mean pulmonary arterial pressure remained constant or rose slightly. If still more of the vascular bed were obstructed, the animal soon died. Welch,[4] Plumier,[5] Tigerstedt,[6, 8] and Gerhardt[7] confirmed these results. Scarff[9] measured the pulmonary arterial pressure in dogs during pneumonectomy. Gibbon and co-workers[10] found that there was no significant effect on arterial and venous pressures until more than 60 per cent of the pulmonary artery was occluded. Fineberg and Wiggers[11] studied the diastolic-systolic right ventricular and aortic pressures in dogs. They concluded that compensatory changes occur in the right heart sufficient to prevent changes in the aortic pressure pulses, up to a point where roughly 58 per cent of the pulmonary circulation was occluded.

Long and co-workers[12] found that if in the dog one lung root is suddenly occluded, a rise occurs in the right ventricular pressure which reaches a peak in two to ten minutes and then falls slowly to return to the pre-ligation level in twenty-five to thirty minutes in most cases.

Since the advent of cardiac catheterization in man, the dynamics of the lesser circulation have been studied in patients with a wide variety of various

This work was supported in part by USPHS Grant No. RG 1254(C), and in part by the Douglas Smith Foundation for Medical Research of The University of Chicago.
Received for publication March 12, 1951.
*From the Department of Surgery of The University of Chicago.
**U. S. Public Health Service Research Fellow of the National Institutes of Health.

pulmonary and cardiac disorders. Cournand[13] and co-workers studied pulmonary hemodynamics before and after pneumonectomy. Mendelsohn[14] and co-workers studied the pulmonary arterial pressure during surgery in a group of eleven patients subjected to various chest operations, including pneumonectomy.

Although it is well known that most patients are able to tolerate a pneumonectomy with surprisingly few obvious circulatory changes, a certain number of patients—notably those in the older age group—are not able to withstand the rigors of a pneumonectomy. In some cases this may be due to the failure of the right ventricle to make a satisfactory adjustment to the strain of having the pulmonary vascular bed suddenly reduced by 50 per cent.

It was thought, therefore, that it would be of interest to study the right ventricular pressures in a controlled animal experiment during and after the removal, in stages, of up to 85 per cent of the pulmonary tissue by a combination of pneumonectomy and lobectomy. The question arose as to whether there might be some correlation between the pressure changes and the survival or death of the animal when the pulmonary tissue was reduced to very low levels.

It is the purpose of this paper to report our observations on the right ventricular pressures during operation; (1) in a group of animals undergoing pneumonectomy; (2) in a group undergoing lobectomy or bilobectomy following pneumonectomy; and (3) in a group where the lung tissue was reduced to one or two lobes by three operations. The right ventricular pressures were determined at intervals following operation in most of the surviving dogs. Also systemic pressures, blood oxygen analysis, and hematocrits were studied.

TECHNIQUE

Normal, healthy mongrel dogs weighing from 10 to 14 kilograms were used. They received 45 mg. of morphine as premedication. Under local anesthesia, an 8F cardiac catheter was introduced, in most cases through one of the external jugular veins into the right ventricle, under fluoroscopic control. Occasionally a femoral vein was used. The right ventricle, rather than the pulmonary artery, was chosen for the measurements for several reasons. It was found that it was easier to maintain the position of the catheter in the ventricle rather than in the artery because of the manipulations taking place before and during the operation. Moreover, it is not always easy to place the tip of the catheter in a given portion of the artery; for instance, the catheter might be located in the artery to be divided. Also artefacts are more common in recording pulmonary arterial pressures than ventricular pressures. Very often some disturbance in rhythm occurred as the catheter was being manipulated into the ventricle, but once it was in place, no further disturbance was observed. Difficulty because of having the tip of the catheter in the right ventricle during the operation was not experienced. Because of convenience, heparin 0.5 mg. per c.c. rather than a saline drip was used to keep the catheter open. The total amount of heparin rarely exceeded 5 mg., and this caused no unusual bleeding during the surgery. Measurements were made either with a Hamilton manometer or a Sanborn Electromanometer, using an electrocardiogram apparatus for recording.

Fig. 1 shows an anteroposterior and a lateral roentgenogram of a normal dog with the cardiac catheter in place.

Recordings of heart rate and right ventricular pressures were made in the following order: while the animal was still awake; after induction of ether anesthesia; after insertion of an endotracheal tube and connection with the anesthesia machine; after the chest was opened; after the artery to the lobe or lung to be removed was mobilized; *immediately* after ligation of the artery; after lobectomy or pneumonectomy; after closure of the chest; and at the conclusion of the procedure with the anesthetic discontinued, as may be seen in the tables.

The anesthesia used for the first operation was ether and air given under slight continuous positive pressure. Ether-oxygen was used for the second and third stages, with controlled respiration while the chest was open. With this method, the animals practically always remained in good condition during the operation, even when the left upper lobe was the only remaining lung tissue.

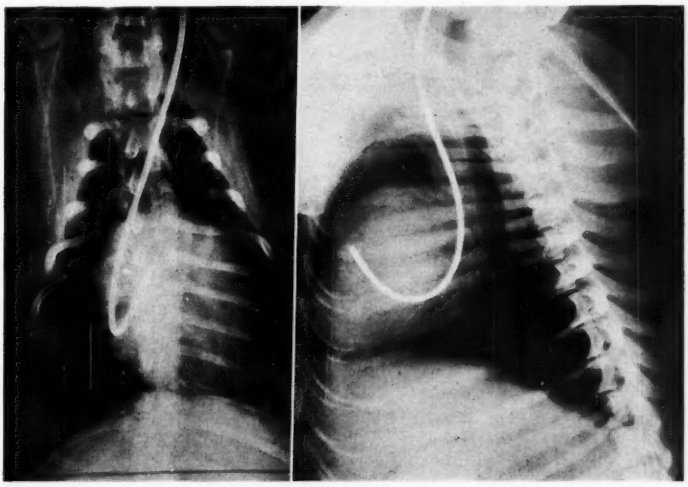


Fig. 1.—Anteroposterior and lateral chest roentgenogram of normal dog showing cardiac catheter in place.

The operations were all done by the usual dissection technique. After completion of the procedure an attempt was made to adjust the intrapleural pressure to about –15 to –5 cm. of water. The opening in the jugular vein was then closed with 00000 deknatel suture to save it for subsequent catheterizations. After the first operation the animal received no special care. After the second or third, the animal was placed in an oxygen box for a few hours. Penicillin was used only exceptionally.

Following the first stage, at least three months were allowed to elapse before further reduction of the pulmonary tissue was carried out in order that the animal might recover fully. In one group of animals, measurements were

TABLE I. RIGHT VENTRICULAR PRESSURES IN MM. OF HG
LEFT OR RIGHT PNEUMONECTOMY

DOG NO.	DOG AWAKE	DOG ASLEEP	POSITIVE PRESSURE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER PNEUMONECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	LIVING OR DEAD
4	40/3	43/0	42/2	41/3	---	45/7	50/7	47/3	43/3	L
36	30/3	34/2	29/0	30/0	33/–2	50/2	50/1	44/12	40/11	L
242	40/1	38/–2	35/2	34/4	43/7	54/10	53/8	45/5	45/2	D
379	33/4	30/4	32/3	35/10	37/3	54/5	56/3	62/2	64/1	L
435	26/2	32/0	36/5	34/5	32/4	42/5	57/3	51/3	51/3	L
436	35/7	33/18	37/22	35/8	37/10	47/11	52/9	57/11	54/8	L
437	34/10	37/6	32/8	30/7	33/8	39/9	58/6	36/5	43/4	L
468	---	---	50/10	42/7	---	--	---	---	42/0	L
494	---	20/1	31/–2	44/3	---	40/10	48/7	57/7	30/3	D
539	40/5	35/5	37/5	38/2	48/4	68/8	54/10	52/12	48/–2	L
865	26/10	30/11	35/2	32/4	35/6	40/6	40/5	40/6	39/6	L
923	25/5	---	28/3	25/0	25/–2	26/–6	30/0	25/10	---	L
924	---	32/6	30/3	32/7	30/4	39/5	48/8	39/2	33/4	L
925	25/5	26/6	---	28/5	32/3	40/10	42/11	---	---	L
985	25/1	22/–4	22/4	26/8	25/8	31/11	43/8	42/8	38/2	D
Av.	32/5	32/4	37/5	34/5	34/4	45/7	49/6	46/7	44/4	

TABLE II. RIGHT VENTRICULAR PRESSURES IN MM. HG; RIGHT LOWER AND ACCESSORY LOBECTOMY IN DOGS HAVING PREVIOUS LEFT PNEUMONECTOMY

DOG NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER LOBECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
9	42/4	45/2	44/2	42/5	44/8	64/12	68/9	78/2	78/0	--	---	D
69	33/8	38/2	42/0	42/0	43/1	75/5	85/8	75/0	75/2	--	---	D
468	---	29/8	30/10	55/10	---	76/10	68/9	47/8	41/5	--	---	D
478	42/3	37/2	25/2	34/4	33/5	38/8	48/4	42/4	42/6	--	---	D
539	58/6	48/3	46/4	48/4	48/5	62/6	80/5	72/2	71/3	--	---	D
617	23/2	29/4	26/6	27/8	28/7	48/8	58/8	50/2	52/2	--	---	D
673	---	30/3	31/5	26/6	36/8	57/12	59/17	44/21	36/12	--	---	L
810	36/2	42/16	36/2	38/5	31/0	50/5	58/5	52/0	51/0	6	36/16	L
855	44/5	46/-2	32/0	30/-2	32/4	38/3	38/2	38/0	35/0	34	60/3	L
857	38/11	35/8	37/3	36/5	38/5	55/6	54/5	58/5	62/4	5	41/5	L
										14	44/8	
901	43/7	34/6	43/4	19/5	31/10	46/17	43/14	62/7	64/6	37	48/0	L
911	34/1	35/1	34/0	30/2	28/3	32/6	58/6	60/3	60/4	--	---	D
924	44/6	38/5	41/5	37/6	39/10	77/10	85/7	90/13	92/12	2	74/3	L
										30	81/7	
925	32/4	38/6	34/3	32/10	37/8	55/11	52/8	68/2	72/1	2	45/3	L
										6	42/-2	
										30	55/4	
936	37/4	28/2	35/0	32/1	36/0	61/2	61/4	44/1	50/0	38	43/10	L
Av.	40/5	37/5	36/3	35/5	36/6	56/8	61/7	59/5	59/4			

taken during each stage; in another group, only during the second or second and third operations.

Fig. 2 shows a bronchogram of the remaining lung tissue in a dog that had undergone pneumonectomy and right lower and accessory lobectomy.

RESULTS

Table I shows the measurements which were taken before, during, and after pneumonectomy in a group of fifteen animals. All animals survived the procedure, three dying later of miscellaneous causes. Nine of these have undergone further reduction in lung tissue. It can be seen that most of the dogs followed the average of the group rather closely. Up to the point just prior to ligation of the artery there was very little change from the initial pressures. For some unexplained reason, Dog 436 had temporarily a very high diastolic pressure. At the point where the artery was ligated, it can be seen that there was a slight to moderate rise in the systolic component, averaging 10 mm. Hg.

A. *B.*

Fig. 2.—*A*, Bronchogram showing entire chest of animal having only the right upper and middle lobes remaining. *B*, Spot film of same animal. 1. Stump of left main bronchus. 2. Stump of bronchus transected above lower and accessory lobe bronchi. 3. Right middle lobe. 4. Right upper lobe.

The systolic pressure was still a little higher in most of the dogs by the time removal of the lung was completed. At the end of the procedure the systolic pressure was somewhat lower in most of the dogs, although it still averaged 9 mm. higher than initially. There was little change in the diastolic pressure in this group during the entire procedure.

Table II shows the measurements of fifteen animals which were taken at the time of removal of the right lower and accessory lobes. All these animals had had a previous left pneumonectomy. All animals survived the operation,

four died within twenty-four hours, and three others between one week and several months later of causes other than cardiorespiratory failure. Eight are still living for periods up to eighteen months. The initial systolic pressure was moderately elevated in several of the animals. As with the pneumonectomy group, however, there was, with a few exceptions, no great change in pressure until ligation of the artery. When the artery was ligated, an *immediate* average rise of 20 mm. Hg occurred in the systolic pressure, with a variation of from 4 mm. to 38 mm. Hg. After the removal of the lung, the average systolic pressure was 5 mm. higher. Little change in pressure was observed from then on until after the anesthetic was stopped. Several of the dogs exhibited a high diastolic pressure at some period during the surgery, but the diastolic pressure for the group remained almost constant throughout.

Table III shows the measurements which were taken at the time of removal of the left lower lobe in eight animals. These dogs had all had the right lung removed previously. Four of these animals died within twenty-four hours of pulmonary edema, two died a few days later of causes not directly related to the small amount of lung tissue remaining, and two are still surviving. These dogs consistently had, prior to ligation of the artery, a higher systolic pressure than the group of animals in Table II. After ligation of the artery the systolic pressure rose an average of 14 mm. Hg, with a variation between 6 and 28 mm. Hg. The systolic pressure rose an average of 8 mm. Hg more by the time the lobe was removed, and had fallen 3 mm. by the time the anesthesia was discontinued. It may be noted that the diastolic pressure was high in this group after ligation of the artery, and remained elevated at the conclusion of the procedure.

Table IV shows the measurements of five animals, four in which the lung tissue was reduced to the left upper lobe, and one to the right upper and middle lobes, by three operations. It was thought that there might be a lower mortality rate with a more gradual reduction in pulmonary tissue. The first four animals all died, two within twenty-four hours, and two later. As the results from these few animals did not seem to indicate any advantage in the three-stage procedure, it was abandoned.

Measurements taken at intervals following surgery in the survivors show right ventricular systolic pressure to be lower than at the end of the operation in most of the animals, but higher than in normal dogs. In several it is very high.

The results of systemic pressures taken before and after surgery showed that the pressure in most cases was not greatly different after the surgery than initially. In general, it tended to be a little higher; significantly lower values were found in none.

The hematocrits taken before, during, and after the procedures did not show any significant change. Except for the fact that the heart rate always rose after induction of anesthesia, no constant changes were noted.

In the surviving animals blood oxygen analyses have shown a normal value in almost every case.

TABLE III. RIGHT VENTRICULAR PRESSURES IN MM. HG; LEFT LOWER LOBECTOMY IN DOGS HAVING PREVIOUS RIGHT PNEUMONECTOMY

DOG NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER LOBECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
4	---	40/1	43/6	44/5	45/8	52/12	58/18	54/8	55/8	--	---	D
36	43/2	45/–2	50/–2	42/4	51/3	80/9	56/9	80/2	80/0	5	43/–2	L
										30	70/6	
379	48/4	41/5	44/6	46/12	48/12	56/15	52/6	55/6	60/9	--	--	D
394	48/8	---	48/4	42/5	48/8	76/15	86/18	73/10	74/12	--	--	D
436	48/4	34/17	40/6	34/12	41/12	54/16	90/11	70/16	63/8	--	--	D
437	53/11	58/10	52/5	38/10	36/11	42/14	72/11	63/8	64/8	--	--	D
486	63/8	59/7	53/6	43/15	46/8	60/9	63/8	68/6	63/8	--	--	L
593	---	42/2	41/5	45/6	42/5	53/8	55/8	52/9	53/8	--	--	D
Av.	50/6	46/7	46/5	42/9	45/8	59/12	67/11	64/8	64/8			

TABLE IV. RIGHT VENTRICULAR PRESSURES IN MM. HG; REDUCTION OF LUNG TISSUE IN THREE STAGES

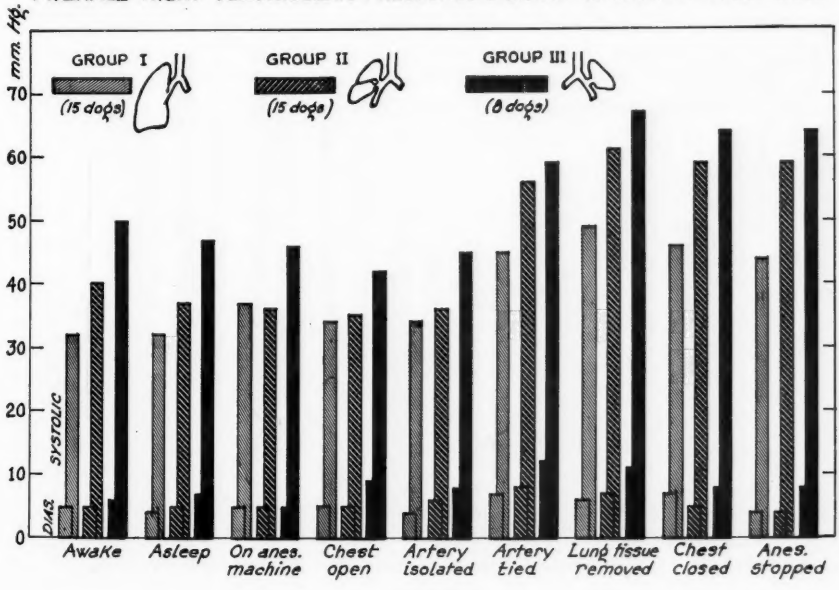
DOG NO.	STAGE NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER OPERATION	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
256	II	---	48/22	40/4	40/10	56/10	76/10	48/20	48/5	48/4	---	---	
	III	44/10	52/7	38/9	35/12	54/10	53/10	67/18	---	---	---	---	D
680	II	---	30/1	28/4	30/3	33/3	36/5	38/8	34/18	30/14	---	---	
	III	56/8	42/10	42/7	40/14	40/16	46/20	46/24	47/20	48/24	40	60/4	L
26	II	35/11	35/10	30/12	30/14	36/17	40/20	44/2	48/20	44/18	---	---	
	III	60/4	---	64/13	74/9	66/20	90/18	120/42	74/10	71/10	---	---	D
104	I	46/3	46/2	40/0	44/3	47/5	53/4	54/10	58/8	47/5	---	---	
	II	32/0	48/7	28/2	23/0	20/0	36/0	34/1	40/0	38/0	---	---	
	III	40/0	38/–2	36/0	34/12	50/7	60/8	58/8	78/9	74/8	---	---	D
999	I	26/3	27/12	32/10	29/13	34/17	38/1	42/5	40/5	38/2	---	---	
	II	55/6	---	72/10	55/11	56/10	78/12	---	---	---	---	---	
	III	48/2	36/0	46/1	56/8	62/5	65/3	72/4	68/10	74/11	---	---	D

DISCUSSION

This approach to the problem of the changes occurring in the lesser circulation when a portion of the pulmonary vascular bed is occluded is different from most of the previous work; lung tissue was removed, and the animals kept alive. Previous work has largely consisted of acute experiments with partial or complete obstruction of the pulmonary artery being carried out over periods varying from a few minutes to several hours. We realize, of course, that reducing the lung tissue in stages gives the heart and the remaining pulmonary vascular bed time for adjustment. Sudden occlusion of all the arteries except to one lobe is not the same as gradual reduction as we have carried out. Also, as the mediastinum is not a stable structure in dogs, the remaining lung tissue will expand until it fills the space previously occupied by both lungs.

GRAPH 1

AVERAGE RIGHT VENTRICULAR PRESSURES DURING REMOVAL OF LUNG TISSUE



Graph 1.

Whether or not the animal survives the reduction of this amount of lung tissue depends not only on the ability of the right heart to adjust to the increased resistance, but also on whether enough pulmonary tissue remains to supply the respiratory needs of the animal. Of the seventeen deaths occurring in the group of dogs that had a second or third operation, ten occurred within a twenty-four-hour period, seven occurred from a few days to several months later of miscellaneous causes. The ten dogs that died soon after operation all exhibited a similar picture of increasingly severe dyspnea and cyanosis. At autopsy all had a

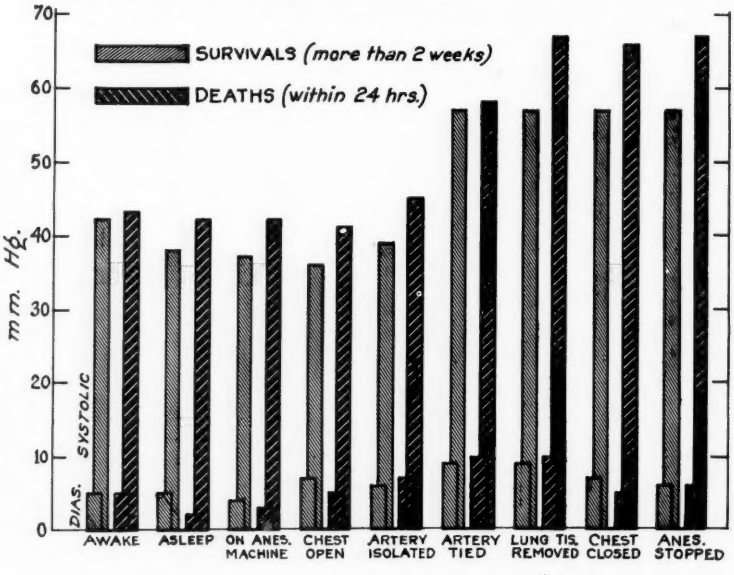
similar prominent finding—massive pulmonary edema. Gross dilatation of the heart was observed in several of these animals, but not all.

It is difficult to separate the cardiac from the pulmonary factors in these animals. However, as soon as pulmonary edema appears, a vicious circle is set up. The remaining lung tissue is less able to oxygenate the blood, causing increasing damage to the pulmonary tissue and myocardium with more pulmonary edema until the animal dies.

The surviving animals all appear surprisingly healthy, although several of them have a very high right ventricular systolic pressure, indicating that the increased resistance caused by the decreased vascular bed has imposed considerable strain on the right ventricle. It would appear that the right ventricle has a considerable ability to compensate; that is, it is able to overcome the increased resistance without any obvious ill effects, at least for many months.

GRAPH 2

AVERAGE RIGHT VENTRICULAR PRESSURES IN SURVIVALS AND DEATHS



Graph 2.

One sees from Graph 1 that a definite difference existed in the systolic pressures in the three groups of animals, which goes along with the amount of reduction in the pulmonary vascular bed. It is not clear just why there was the observed difference between groups 2 and 3 up to the point of ligation of the artery. Up to this point the only difference in the amount of reduction was that the dogs in group 2 had a left pneumonectomy, while those in group 3 had a right pneumonectomy. Although the right lung is somewhat larger than the left, it is questionable whether this is the only factor accounting for the observed

difference. After ligation the amount of increase in pressure would appear to correlate rather well with the amount of reduction of the vascular bed. The diastolic pressure in group 3 was well above that in group 2, especially after ligation of the artery, being considerably above normal in many of the dogs in group 3. This may indicate that the right heart was very nearly on the verge of decompensation, when this amount of reduction was carried out.

From this graph, it is apparent that at several points the animals in group 1 had slightly higher pressures than those in group 2. The fact that group 1 had continuous positive pressure anesthesia might account for this.

From Graph 2, which shows the average pressures of the dogs dying within twenty-four hours and those surviving the last operation by at least two weeks, one gets the impression that the dogs which died had somewhat higher pressures than those that survived. However, the individual variations are such that this is probably of little significance.

It is apparent from the tables that the mortality in the group having only the left upper lobe remaining was much higher than in the group where the right upper and middle lobes were the only remaining lung tissue. It is probable that the difference in the amount of remaining lung tissue in the two groups accounted for this. It is also probable that in most dogs the point of maximum reduction compatible with life lies just between these two groups.

The follow-up pressures in the surviving animals indicate, as one might expect, that in most cases the systolic pressure falls as the remaining pulmonary vascular bed becomes adjusted to the increased blood flow through it. However, most of the pressures remain at levels higher than normal, indicating an increased resistance to the right heart output.

SUMMARY AND CONCLUSIONS

1. Data are presented on changes occurring in the right ventricular pressure in dogs during removal of lung tissue.

2. In the majority of animals decreasing the pulmonary tissue caused a rise in the right ventricular systolic pressure somewhat in proportion to the amount removed.

3. Many dogs tolerated reduction to the right middle and upper lobes. Very few were able to tolerate removal of all pulmonary tissue except the left upper lobe. If the animal survived the first twenty-four hours following operation, the chance of prolonged survival was good.

4. It was difficult to correlate the pressure changes with the survival or death of the animal.

5. The right ventricle in dogs has considerable power of compensation, providing the stress is imposed gradually.

REFERENCES

1. Beutner, E.: Ueber die Strom-und Druckkräfte des Blutes in der Arteria und Vena pulmonalis, Zeitschrift für Rationelle Medicin, Neue Folge II Bd., 1852.
2. Badoud, E.: Ueber den Einflues des Hirns auf den Druck in der Lungen-arterie, Verhandlungen der Würzburger physicalisch-medicinischen Gesellschaft, Neue Folge VIII Bd., 1874.

3. Lichtheim, Ludwig: Die Störungen des Lungenkreislaufs und ihr Einfluss auf den Blutdruck, Inaug. Dissert Breslau, 1876.
4. Welch, W. H.: Zur Pathologie des Lungenodems, Virchows Archiv. f. path. Anat. **72:** 375, 1878.
5. Plumier, L.: La Circulation Pulmonaire Chez le Chien, Arch. Internat. de Physiologie, Vol. 1, 1904, p. 176.
6. Tigerstedt, R.: Ueber den Lungenkreislauf, Skandinav. Archiv. für Physiologie **14:** 259, 1903.
7. Gerhardt, D.: Experimentelle Beitrage zur Lehr von Lungenkreislauf, etc., Ztschr. f. klin. Med. **55:** 195, 1904.
8. Tigerstedt, Robert: Die physiologie Des Kreis laufes, de Gruyter, Leipzig, 1923.
9. Scarff, J. E.: Pulmonary Blood Pressures, Arch. Surg. **12:** 591, 1926.
10. Gibbon, J. H., Hopkinson, M. and Churchill, E. D.: Changes in the Circulation Produced by Gradual Occlusion of the Pulmonary Artery, J. Clin. Investigation **11:** 543, 1932.
11. Fineberg, M. D., and Wiggers, C. J.: Compensation and Failure of the Right Ventricle, Am. Heart J. **11:** 255, 1936.
12. Long, J. H., Wester, M. R., Oppenheimer, M. J.: Immediate Cardiovascular and Respiratory Adjustments to Pneumonectomy, J. THORACIC SURG. **18:** 268-278, 1949.
13. Cournand, A., Riley, R. L., Himmelstein, A., and Austrian, R.: Pulmonary Circulation and Alveolar Ventilation Perfusion Relationships after Pneumonectomy, J. THORACIC SURG. **19:** 80, 1949.
14. Mendelsohn, Harvey J., Zimmerman, Henry A., and Adelman, Arthur: A Study of Pulmonary Hemodynamics During Pulmonary Resection, J. THORACIC SURG. **20:** 366-372, Sept., 1950.

The American Association for Thoracic Surgery

1951-1952

Officers

PRESIDENT, FRANK B. BERRY, New York, N. Y.
VICE-PRESIDENT, ROBERT M. JANES, Toronto, Ontario
TREASURER, WILLIAM E. ADAMS, Chicago, Ill.
SECRETARY, PAUL C. SAMSON, Oakland, Calif.
EDITOR, EVARTS A. GRAHAM, St. Louis, Mo.

Council

ROBERT R. SHAW
CAMERON HAIGHT
EMILE HOLMAN
JOHN H. GIBBON, JR.
ALFRED BLALOCK

Membership Committee

CHAIRMAN, EDWARD M. KENT
O. T. CLAGETT
LYMAN A. BREWER
JOHN M. CHAMBERLAIN
ERNEST C. JANES

LIST OF MEMBERS OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY, 1951-1952

Honorary Members

BROCK, MR. R. C.---Guy's Hospital, London, England
CRAFOORD, CLARENCE---Sabbatsberg sjukhus, Stockholm, Sweden
DAVIES, H. MORRISTON---Ruthin, North Wales, G. B.
DENK, WOLFGANG---Surgical University Clinic, Vienna, Austria
GREEN, NATHAN W.---New Canaan, Conn.
JACKSON, CHEVALIER---Old Sunrise Mills, Schwenkville, Pa.
KELLER, WILLIAM L., COL. M.C., USA, RET'D---2930 Foxhall Rd., N.W., Washington, D. C.
LERCHE, WILLIAM---Larkhills, Cable, Wis.
MATAS, RUDOLPH---2255 St. Charles Ave., New Orleans, La.
NEMB, CARL---Ullevaal Hospital, Oslo, Norway
SHENSTONE, NORMAN S.---904 Medical Arts Bldg., Toronto 5, Ontario

Active Members

ABBOTT, OSLER---516 E. Wesley Road, N.E., Atlanta, Ga.
ADA, ALEXANDER E. W.---139 E. 94th St., New York, N. Y.
ADAMS, HERBERT D.---Lahey Clinic, 605 Commonwealth Ave., Boston, Mass.
ADAMS, RALPH---Good Samaritan Hospital, Woodberry, Tenn.
ADAMS, WILLIAM E.---University of Chicago, Chicago 27, Ill.
ALEXANDER, JOHN---University Hospital, Ann Arbor, Mich.
ALLBRITTEN, FRANK F., JR.---1025 Walnut St., Philadelphia, Pa.
AUFSES, ARTHUR H.---1155 Park Ave., New York, N. Y.

BALLON, HARRY C.---1414 Drummond, St., Montreal, Quebec
BARKLEY, HOWARD THOMAS---3103 Fannin St., Houston 4, Texas
BARNWELL, JOHN B.---Room 866, Veterans Administration, Washington, D. C.
BEATTIE, EDWARD JAMES, JR.---George Washington University Hospital, 901 23rd St. N. W., Washington, D. C.
BECK, CLAUDE S.---2065 Adelbert Road, Cleveland, Ohio
BENSON, CLIFFORD D.---307 David Whitney Bldg., Detroit, Mich.
BETTS, REEVE HAWKINS---1101 Beacon St., Brookline, Mass.
BIRD, CLARENCE---21 John Street, Providence, R. I.
BISGARD, DEWEY---1420 Medical Arts Bldg., Omaha, Neb.
BLADES, BRIAN---George Washington University Hospital, 901 23rd Street, N. W., Washington, D. C.
BLALOCK, ALFRED---Johns Hopkins Hospital, Baltimore, Md.
BLOCK, ROBERT G.---University of Chicago, Chicago, Ill.
BRADSHAW, HOWARD---Wake Forest College, Bowman Gray School of Medicine, Winston-Salem, N. C.
BRANTIGAN, OTTO C.---104 W. Madison St., Baltimore, Md.
BREWER, LYMAN AUGUSTUS---Suite 1400, 1930 Wilshire Blvd., Los Angeles, Calif.
BROWN, A. LINCOLN---490 Post St., San Francisco, Calif.
BUCKINGHAM, WILLIAM W.---314 Professional Bldg., Kansas City, Mo.
BUGDEN, WALTER F.---Medical Arts Bldg., Syracuse, N. Y.
BURFORD, THOMAS H.---Barnes Hospital, St. Louis, Mo.
BURNETT, W. EMORY---Temple University Hospital, Broad and Ontario Sts., Philadelphia, Pa.
BYRON, FRANCIS R. X.---Wadsworth General Hospital, Los Angeles, Calif.

CARR, DUANE—899 Madison Ave., Memphis, Tenn.
CARTER, B. NOLAND—Cincinnati General Hospital, Cincinnati, Ohio
CHAMBERLAIN, JOHN MAXWELL—139 E. 36th St., New York 16, N. Y.
CHURCHILL, EDWARD D.—Massachusetts General Hospital, Boston, Mass.
CLAGETT, O. THERON—Mayo Clinic, Rochester, Minn.
COHN, ROY BARNETT—Stanford University Hospital, San Francisco, Calif.
COLEMAN, FRANK PHILIP—810 W. Franklin St., Richmond, Va.
COURNAND, ANDRE—Bellevue Hospital, 28th St. and 1st Ave., New York, N. Y.
CRIMM, PAUL D.—Beohne Tuberculosis Hospital, Evansville, Ind.
CURRERI, ANTHONY R.—1300 University Ave., Madison, Wis.
CURTIS, GEORGE M.—Ohio State University, College of Medicine, Columbus, Ohio

DANIEL, ROLLIN A.—Vanderbilt University Medical School, Nashville, Tenn.
DAVIDSON, LOUIS R.—5 E. 53rd St., New York, N. Y.
DAVIS, EDGAR W.—1150 Connecticut Ave., Washington, D. C.
DAY, J. CLAUDE—307 David Whitney Bldg., Detroit 26, Mich.
DEBAKEY, MICHAEL E.—Baylor School of Medicine, Department of Surgery, Houston, Texas
DECKER, HARRY R.—1010 Professional Bldg., 429 Penn Ave., Pittsburgh, Pa.
DOLLEY, FRANK S.—1930 Wilshire Blvd., Los Angeles 5, Calif.
DORNER, RALPH A.—710 Equitable Bldg., Des Moines 9, Iowa
DORSEY, JOHN M.—55 E. Washington St., Chicago 2, Ill.
DOUGLASS, RICHMOND—Biggs Memorial Hospital, Ithaca, N. Y.
DRASH, EVERETT C.—University of Virginia Hospital, Charlottesville, Va.
DUGAN, DAVID J.—2938 McClure St., Oakland, Calif.

EFFLER, DONALD B.—Euclid and E. 93rd St., Cleveland, Ohio
EHLER, ADRIAN A.—Albany Hospital, Albany, N. Y.
ELKIN, DANIEL C.—Emory University Hospital, Atlanta, Ga.

FLICK, JOHN B.—The Pennsylvania Hospital, 8th and Spruce Sts., Philadelphia, Pa.
FORSEE, JAMES H., COL. M.C., U.S.A.—Fitzsimons General Hospital, Denver, Colo.
FREEDLANDER, SAMUEL O.—10515 Carnegie Ave., Cleveland, Ohio

GALE, JOSEPH W.—Wisconsin General Hospital, Madison 6, Wis.
GARLOCK, JOHN H.—50 E. 77th St., New York, N. Y.
GEBAUER, PAUL—Leahi Hospital, 649 Pokole Street, Honolulu, T. H.
GIBBON, JOHN H., JR.—Jefferson Medical College, 1025 Walnut St., Philadelphia, Pa.
GRACE, ARCHIBALD J.—530 Wellington St., London, Ontario
GRAHAM, EVARTS A.—Barnes Hospital, St. Louis 10, Mo.
GRAY, HOWARD K.—Mayo Clinic, Rochester, Minn.
GROSS, ROBERT E.—300 Longwood Ave., Brookline, Mass.
GROW, JOHN B.—3705 E. Colfax, Denver, Colo.

HAIGHT, CAMERON—University Hospital, Ann Arbor, Mich.
HANLON, ROLLINS—St. Louis University School of Medicine, 1402 S. Grand Blvd., St. Louis, Mo.
HARKEN, DWIGHT E.—314 Commonwealth Ave., Boston, Mass.
HARPER, FREDERICK R.—1104 Republic Bldg., Denver, Colo.
HARRISON, HARLON W.—U. S. Veterans Hospital, San Fernando, Calif.
HART, DERYL—Duke University, Durham, N. C.
HARTER, JOHN S.—1010 Heyburn Bldg., Louisville 2, Ky.
HEAD, JEROME R.—55 E. Washington St., Chicago, Ill.
HIMMELSTEIN, AARON—70 E. 96th St., New York 28, N. Y.

HOLINGER, PAUL H.----700 N. Michigan Ave., Chicago, Ill.
HOLMAN, EMILE----722 Funston St., San Francisco, Calif.
HOLMAN, CRANSTON WILLIAM----525 E. 68th St., New York, N. Y.
HUDSON, W. A.----602 David Whitney Bldg., Detroit, Mich.
HUMPHREYS, GEORGE H.----180 Ft. Washington Ave., New York, N. Y.

JACKSON, CHEVALIER L.----Temple University Hospital, 3401 N. Broad St., Philadelphia, Pa.
JANES, ERNEST C.----250 Main St., Hamilton, Ontario
JANES, ROBERT M.----Medical Arts Bldg., Toronto, Ontario
JOANNIDES, MINAS----4753 Broadway, Chicago, Ill.
JOHNSON, JULIAN----University of Pennsylvania Hospital, Philadelphia, Pa.
JONES, JOHN C.----1136 W. Sixth St., Los Angeles, Calif.

KAY, EARLE B.----10465 Carnegie Ave., Cleveland, Ohio
KENT, EDWARD M.----1107 Clark Bldg., Pittsburgh 22, Pa.
KERGIN, FREDERICK G.----Medical Arts Bldg., Toronto 5, Ontario
KINSELLA, THOMAS J.----1251 Medical Arts Bldg., Minneapolis, Minn.
KNOEPP, LOUIS F.----Veterans Administration Hospital, Alexandria, La.

LAIRD, ROBERT----399 Bathhurst St., Toronto Western Hospital, Toronto, Ontario
LAM, CONRAD R.----Henry Ford Hospital, Detroit, Mich.
LANGSTON, HIRAM THOMAS----307 David Whitney Bldg., Detroit 26, Mich.
LEAHY, LEON J.----105 Medical Arts Bldg., Buffalo, N. Y.
LESTER, CHARLES W.----70 E. 80th St., New York, N. Y.
LEVEN, N. LOGAN----1464 Lowry Medical Arts Bldg., St. Paul, Minn.
LINDSKOG, GUSTAF E.----50 Marvel Road, New Haven, Conn.
LYNCH, JOSEPH P.----1180 Beacon St., Brookline, Mass.

MCGRATH, EDWARD J.----Cincinnati General Hospital, Cincinnati, Ohio
MCINTOSH, CLARENCE A.----1390 Sherbrooke St., W., Montreal, Quebec
MAIER, HERBERT C.----3 E. 71st St., New York, N. Y.
MAJOR, ROBERT CARLISLE----University Hospital, Augusta, Ga.
MAUTZ, F. R.----University Hospital, Cleveland, Ohio
MEADE, RICHARD H., JR.----Blodgett Medical Bldg., Grand Rapids, Mich.
MELTZER, HERBERT----Medical Director, Charles Camsell Hospital, Edmonton, Alta.
MERKEL, CARL G.----8 Church St., Saranac Lake, N. Y.
MEYER, HERBERT WILLY----170 E. 78th St., New York, N. Y.
MISCALL, LAURENCE----11 E. 68th St., New York, N. Y.
MOERSCH, HERMAN----725 Tenth Avenue, Rochester, Minn.
MOORE, JULIAN A.----404 Flatiron Bldg., Asheville, N. C.
MOORE, RICHMOND L.----180 Ft. Washington Ave., New York, N. Y.
MUDD, JAMES L.----634 N. Grand Blvd., St. Louis, Mo.
MULVIHILL, DANIEL A.----15 E. 77th St., New York, N. Y.
MURPHY, JAMES D.----Veterans Administration Hospital, Oteen, N. C.

NEHIL, LAWRENCE W.----709 Hume Mansur Bldg., Indianapolis, Ind.
NEWTON, HARLAN F.----319 Longwood Ave., Boston, Mass.
NIXON, J. W.----1121 Nix Professional Bldg., San Antonio, Texas

O'BRIEN, EDWARD J.----307 David Whitney Bldg., Detroit 26, Mich.
OCHSNER, ALTON----Tulane University School of Medicine, Department of Surgery, New Orleans, La.
O'ROURKE, PAUL V.----307 David Whitney Bldg., Detroit, Mich.
OVERHOLT, RICHARD H.----1101 Beacon St., Brookline, Mass.

PAINE, JOHN RANDOLPH—The Buffalo General Hospital, 100 High St., Buffalo, N. Y.
PAULSON, DONALD L.—3810-12 Swiss Ave., Dallas, Texas
POOL, JOHN LAWRENCE—140 E. 54th St., New York, N. Y.
POTTS, WILLIS J.—707 Fullerton Ave., Chicago 14, Ill.
PROCTOR, OSCAR C.—Medical-Dental Bldg., Seattle, Wash.

RAINE, FORRESTER—425 E. Wisconsin Ave., Milwaukee, Wis.
RIENHOFF, WILLIAM F., JR.—1201 N. Calvert St., Baltimore, Md.
RIGGINS, H. MCLEOD—140 E. 54th St., New York, N. Y.
ROBERTSON, ROSS—3830 West 37th Ave., Vancouver, B. C.
ROGERS, W. L.—490 Post Street, San Francisco, Calif.
ROSEMOND, GEORGE PARROTT—3401 Broad St., N., Philadelphia, Pa.
ROSS, DUDLEY E.—974 Dunsmuir Road, Mount Royal, Quebec
RUMEL, WILLIAM R.—807 Medical Arts Bldg., Salt Lake City, Utah

SAMSON, PAUL C.—2938 McClure St., Oakland, Calif.
SANGER, PAUL W.—1518 Harding Place, Charlotte, N. C.
SCANNELL, J. GORDON—Massachusetts General Hospital, Boston, Mass.
SCHAFFNER, VERNON D.—Kentville, N. S.
SHAW, ROBERT R.—3810-12 Swiss Ave., Dallas, Texas
SHEFTS, LAURENCE M.—503 Moore Bldg., San Antonio, Texas
SKINNER, GEORGE F.—36 Coburg St., St. John, N. B.
SOMMER, GEORGE N. J., JR.—120 W. State St., Trenton, N. J.
STEELE, J. D.—1705 W. Wisconsin Ave., Milwaukee, Wis.
STEPHENS, H. BRODIE—384 Post St., San Francisco, Calif.
STRIEDER, JOHN W.—1180 Beacon Street, Brookline, Mass.
SWEET, RICHARD HARWOOD—205 Beacon Street, Boston, Mass.

THOMPSON, SAMUEL A.—850 Park Ave., New York, N. Y.
TOUROFF, ARTHUR S. W.—994 Fifth Ave., New York 28, N. Y.
TUTTLE, WILLIAM M.—307 David Whitney Bldg., Detroit 26, Mich.
TYSON, M. DAWSON—Hitchcock Clinic, Hanover, N. H.

VAN HAZEL, WILLARD—224 S. Michigan Blvd., Chicago, Ill.
VARCO, RICHARD L.—Dept. of Surgery, University of Minnesota, University Hospital, Minneapolis, Minn.
VINEBERG, ARTHUR M.—1108 Elgin Terrace, Apt. 101, Pell St., Montreal, Quebec

WANGENSTEEN, OWEN H.—University Hospital, Minneapolis, Minn.
WATERMAN, DAVID H.—607 Medical Arts Bldg., Knoxville, Tenn.
WATSON, WILLIAM LAW—1088 Park Ave., New York, N. Y.
WEINBERG, JOSEPH A.—Veterans Administration Hospital, Long Beach, Calif.
WELLES, EDWARD S.—110 Park Ave., Saranac Lake, N. Y.
WHITE, MARION LAWRENCE, JR.—First Huntington Nat. Bank Bldg., Huntington, W. Va.
WILLIAMS, MARK H.—63 Frank St., Binghamton, N. Y.
WILSON, NORMAN J.—1105 Beacon St., Brookline, Mass.
WOODRUFF, WARRINER—8 Church St., Saranac Lake, N. Y.
WOODS, FRANCIS M.—1101 Beacon St., Brookline, Mass.
WYLIE, ROBERT H.—535 Park Ave., New York, N. Y.

Associate Members

ACKMAN, F. DOUGLAS—1374 Sherbrooke St., W., Montreal, Quebec
AITCHISON, DAVID BANCROFT—Mount Sanatorium, Hamilton, Ontario
AUERBACH, OSCAR—121 Hamden Ave., Staten Island, N. Y.

BADGER, THEODORE I.----264 Beacon St., Boston, Mass.
BAILEY, CHARLES P.----218 Runnymede Ave., Jenkintown, Pa.
BENEDICT, EDWARD BENSON----Massachusetts General Hospital, Boston, Mass.
BORTONE, FRANK----2765 Hudson Blvd., Jersey City, N. J.
BOYD, DAVID P.----605 Commonwealth Ave., Boston, Mass.
BRINDLEY, GEORGE VALTER, JR.----Scott and White Clinic, Temple, Texas
BROWN, ROBERT K.----806 Metropolitan Bldg., Denver, Colo.
BURBANK, BENJAMIN----142 Jorslemon St., Brooklyn 2, N. Y.

CARSWELL, JAMES, JR.----Veterans Administration Hospital, McKinney, Texas
CHANDLER, JOHN HUGHES----420 E. Main St., Jackson, Tenn.
CHODOFF, RICHARD JOSEPH----1823 Pine St., Philadelphia, Pa.
CHUNN, CHARLES FRANCIS----442 W. Lafayette St., Tampa 6, Fla.
CHURCHILL, AMBROSE SEVIER----(Mail Returned) 806 Medico-Dental Bldg., San Diego, Calif.
CONDON, WILLIAM B.----1008 Republic Bldg., Denver, Colo.
CONKLIN, WILLIAM S.----2718 W. Fairview Blvd., Portland, Ore.
COOPER, DAVID A.----1520 Spruce St., Philadelphia, Pa.
COTTON, BERT HOLLIS----120 S. Lasky Drive, Beverly Hills, Calif.
COX, WILLIAM V.----133 Court St., Auburn, Maine
CRACOVANER, ARTHUR J.----114 E. 72nd St., New York, N. Y.
CRANDELL, WALTER B.----Veterans Administration Hospital, White River Junction, Vt.
CRECCA, ANTHONY DANIEL----376 Roseville Ave., Newark 7, N. J.

DAILEY, JAMES E.----3214 Reba Drive, Houston, Texas
DAVISON, RICHARD----5300 N. Christian Ave., Chicago 13, Ill.
DECAMP, PAUL TRUMBULL----1835 General Pershing, New Orleans, La.
DENNIS, CLARENCE E.----740 Linwood Ave., St. Paul, Minn.
DESHAIES, GEORGES----37 Bellingham Rd., Montreal, Quebec
DETERLING, RALPH A., JR.----180 Ft. Washington Ave., New York, N. Y.
DODRILL, FOREST DEWEY----641 David Whitney Bldg., Detroit, Mich.
DOMM, SHELDON E.----605 W. Main Ave., Knoxville, Tenn.
DRAKE, EMERSON H.----29 Deering St., Portland, Maine

EGLEE, EDWARD P.----105 E. 53rd St., New York, N. Y.
EHRENHAFT, JOHANN L.----University of Iowa, Iowa City, Iowa
ELLISON, ROBERT G.----University Hospital, Augusta, Ga.
EVANS, BYRON HENRY----1930 Wilshire Blvd., Suite 1400, Los Angeles, Calif.

FALOR, WILLIAM H.----623 Second National Bldg., Akron 8, Ohio
FINNEY, GEORGE G.----2947 St. Paul St., Baltimore, Md.
FISCHER, WALTER W.----3 East 71st St., New York 21, N. Y.
FISHBACK, FREDERICK C.----1835 Eye St., N. W., Washington, D. C.
FRANK, HOWARD A.----330 Brookline Ave., Boston 15, Mass.
FRIEDLANDER, RALPH----Veterans Administration Hospital, Brooklyn, N. Y.

GAGNON, EDOUARD DONAT----3411 St. Denis St., Montreal, Quebec
GEARY, PAUL----909 Park Avenue, Plainfield, N. J.
GERBODE, FRANK L.----Stanford University Hospital, San Francisco, Calif.
GLOVER, ROBERT PRENTICE----93 West Levering Mill Road, Bala-Cynwyd, Philadelphia, Pa.
GOLDMAN, ALFRED----Mid Wilshire Medical Bldg., 6317 Wilshire Blvd., Los Angeles, Calif.
GORDON, JOSEPH----New York State Tuberculosis Hospital, Ray Brook, N. Y.
GRACE, EDWIN J.----121 Fort Green Place, Brooklyn, N. Y.
GRISWOLD, R. A.----323 E. Chestnut St., Louisville, Ky.
GUPTILL, PLIMPTON----16 N. Goodman St., Rochester, N. Y.

HARRISON, ALBERT WILSON—Medical Branch, University of Texas, Galveston, Texas
HARRISON, WILLIAM ELLIOTT—710 Seymour St., Vancouver, B. C.
HELMSWORTH, JAMES A.—Cincinnati General Hospital, Cincinnati, Ohio
HERBEN, GEORGE F.—House of Rest at Sprain Ridge, Yonkers, N. Y.
HEROY, WILLIAM W.—22 Oakwood Rd., Huntington, N. Y.
HIGGINSON, JOHN F.—2455 N. W. Marshall St., Portland, Ore.
HOCHBERG, LEW A.—135 Eastern Parkway, Brooklyn 17, N. Y.
HOLLINGSWORTH, RUSSELL K.—Baker V. A. Hospital, Martinsburg, W. Va.
HOPKINS, WILLIAM A.—1374 Villa Drive, Atlanta, Ga.
HUDSON, THEODORE R.—55 E. Washington St., Chicago, Ill.
HUGHES, FELIX A., JR.—Kennedy Hospital, Memphis, Tenn.
HUN, HENRY—149 Washington Ave., Albany, N. Y.
HURLEY, G. A. PATRICK—3001 Kent Ave., Montreal, Quebec
HURWITZ, ALFRED—Veterans Administration Hospital, Newington, Conn.

INGRAM, IVAN N.—909 Hyde St., San Francisco, Calif.
IOVINE, VINCENT M.—1150 Connecticut Ave., N. W., Washington, D. C.

JARVIS, FRED J.—900 Boylston Ave., Seattle, Wash.
JOHNSON, ELGIE K.—909 President St., Brooklyn, N. Y.
JOHNSON, HOLLIS E.—2122 West End Ave., Nashville, Tenn.
JOYNT, G. H. C.—170 St. George St., Toronto, Ontario
JUDD, ARCHIBALD R.—304 N. Fourth St., Hamburg, Pa.

KELLEY, WINFIELD O.—Uncas on Thames, Norwich, Conn.
KESSLER, CHARLES R.—Suite 1027, Woodward Bldg., Birmingham 3, Ala.
KING, CHARLES R.—814 Doctors Bldg., Atlanta, Ga.
KIPP, HAROLD A.—Mercy Hospital, Pittsburgh, Pa.
KIRBY, CHARLES KEELING—3400 Spruce St., Philadelphia 4, Pa.
KLASSEN, KARL PETER—Dept. of Research Surgery, Ohio State University, Columbus, Ohio
KLEPSER, ROY GEORGE—Suite 501, 1835 Eye St., N. W., Washington, D. C.
KLOPSTOCK, ROBERT—Triboro Hospital, Jamaica, N. Y.
KUNDERMAN, PHILLIP JOHN—165 Livingston Ave., New Brunswick, N. J.
KUNSTLER, WALTER E.—5238 Queen Mary Rd., Montreal, Quebec

LAMBERT, ADRIAN, JR.—122 E. 76th St., New York, N. Y.
LAMPSON, RUTLEDGE STARR—85 Jefferson St., Hartford, Conn.
LAUREY, JAMES R.—1318 Girard St., N. W., Washington, D. C.
LAWRENCE, EDWIN A.—Dept. of Surgery, Indiana University Medical Center, Indianapolis, Ind.
LEES, WILLIAM MORRIS—30 N. Michigan, Chicago, Ill.
LEETCH, HENRY W.—108 Main St., Saranac Lake, N. Y.
LEWIS, RUBIN M.—IRO-Area 7, A. P. O. 407, c/o Postmaster, New York, N. Y.
LONGACRE, JACOB JAMES—1503 Carew Tower, Cincinnati, Ohio
LYON, CLAYTON—384 Post St., San Francisco, Calif.

MACDONALD, NEIL—Medical Arts Bldg., Windsor, Ontario
MACKLER, SAUL ALLEN—104 S. Michigan Ave., Chicago 3, Ill.
MACMANUS, JOSEPH—491 Delaware, Buffalo, N. Y.
MACPHERSON, LACHLAN—St. John Tuberculosis Hospital, E. St. John, N. B.
MADER, VICTOR O.—149 S. Park St., Halifax, Nova Scotia, Canada
MASON, JAMES MONROE, III—1023 S. 20th St., Birmingham, Ala.
MAURER, ELMER P. R.—827 Union Central Bldg., Cincinnati 2, Ohio
MAYER, JOHN HENRY, JR.—829 W. 55th St., Kansas City, Mo.
MCDONALD, JOHN R.—Mayo Clinic, Rochester, Minn.
MERENDINO, A. A.—University of Washington, Seattle, Wash.

MICHELSON, ELLIOTT—1801 Eutaw Place, Baltimore, Md.
MIDELFART, PETER A.—321 Summit Ave., Eau Claire, Wis.
MILLER, CARROL CAMERON—205 Beacon St., Boston 15, Mass.
MILLER, FELIX P.—109 N. Oregon St., El Paso, Texas
MINOR, GEORGE R.—University of Virginia Hospital, Charlottesville, Va.
MOUSEL, LLOYD H.—Dept. of Anesthesiology, The Swedish Hospital, Seattle, Wash.

OATWAY, WILLIAM H., JR.—La Vina Sanatorium, La Vina, Calif.
O'NEILL, JAMES FRANCIS—32 Roslyn Ave., Glenside, Pa.
O'NEILL, THOMAS J.—230 N. Broad St., Philadelphia, Pa.

PARKER, EDWARD F.—70 Hasell St., Charleston, S. C.
PHILLIPS, FRANCIS J.—Seward Sanatorium, Seward, Alaska
PINKHAM, ROLAND DAVIS—1106 Cobb Bldg., Seattle, Wash.
POLLOCK, WILLIAM C., COL. M.C., U.S.A.—Fitzsimons General Hospital, Denver, Colo.
POPPE, J. KARL—1130 S. W. Morrison St., Portland, Ore.
POTTER, BENJAMIN P.—Chest Division, Medical Center, Jersey City, N. J.
PRATT, LAWRENCE ARTHUR—Suite 800, Doctors Bldg., 3919 John R. St., Detroit 1, Mich.

RAMSEY, BEATTY H.—10962 Le Conte Ave., Westwood Village, Los Angeles, Calif.
RASMUSSEN, RICHARD ALLEN—Blodgett Medical Bldg., Grand Rapids, Mich.
RAVITCH, MARK M.—Johns Hopkins Hospital, Baltimore, Md.
RICHARDS, VICTOR—Stanford-Lane Hospital, San Francisco 15, Calif.
RYAN, THOMAS C.—U. S. Naval Hospital, St. Albans, N. Y.

SANES, GILMORE M.—530 Medical Arts Bldg., Pittsburgh, Pa.
SCHAFER, PAUL W.—University of Kansas Medical Center, Kansas City, Kan.
SCHMIDT, HERBERT WM.—Mayo Clinic, Rochester, Minn.
SCOTT, HENRY W., JR.—207 Longwood Road, Baltimore, Md.
SEALY, WILL C.—Duke University Hospital, Durham, N. C.
SELEY, GABRIEL PARKUST—39 E. 61st St., New York, N. Y.
SEYBOLD, WILLIAM DEMPSEY—Mayo Clinic, Rochester, Minn.
SHIPMAN, SIDNEY—490 Post St., San Francisco, Calif.
SHUMACKER, HARRIS B., JR.—Indiana University Medical Center, Indianapolis, Ind.
SIMPSON, H. MURRAY—292 Queen's Ave., London, Ontario
SKINNER, A. M.—Homer Folks Tuberculosis Hospital, Oneonta, N. Y.
SKINNER, EDWARD F.—899 Madison Ave., Memphis, Tenn.
SLOAN, HERBERT E.—1225 Kensington Dr., Ann Arbor, Mich.
SNYDER, HOWARD ERROL—103½ E. Ninth Ave., Winfield, Kan.
SOUTTER, LAMAR—205 Beacon St., Boston, Mass.
STENSTROM, JOHN D.—2390 Bowker Ave., Victoria, B. C.
STEWART, ARCHIBALD—722 Drummond Medical Bldg., Montreal, Quebec
STRANAHAN, ALLAN—Albany Hospital, Albany, N. Y.
SULLIVAN, HERBERT J.—58 Wellington St., South, Hamilton, Ontario, Canada
SWAN, HENRY, II—410 Marion St., Denver, Colo.
SWENSON, ORVAR—300 Longwood Ave., Boston, Mass.

TERRILL, FRANK I.—Montana State Tuberculosis Sanatorium, Route 1, Deer Lodge, Montana
TEST, FREDERICK C., II—91 Cass Ave., Mount Clemens, Mich.
THORNTON, THOMAS F., JR.—309 Allen St., Waterloo, Iowa
TILLOU, DONALD J.—311 W. Church St., Elmira, N. Y.

VEAL, J. ROSS—3560 Appleton St., N. W., Washington, D. C.
VORWALD, ARTHUR J.—Director of Research, Trudeau Foundation, 7 Church St., Saranac Lake, N. Y.

WEISEL, WILSON—324 E. Wisconsin Ave., Milwaukee 2, Wis.
WHITESIDE, WILLIAM CARLETON—342 Birks Bldg., Edmonton, Alberta
WILLAUER, GEORGE—1930 Chestnut St., Philadelphia, Pa.
WILSON, JULIUS LANE—Ochsner Clinic, 3503 Prytania St., New Orleans, La.
WIPER, THOMAS B.—536 Mason Street, San Francisco, Calif.
WRIGHT, GEORGE WILBUR—103 Park Ave., Saranac Lake, N. Y.

Senior Members

ALLEN, DUFF S.—Washington University Medical School, St. Louis, Mo.
AMBERSON, J. B.—Bellevue Hospital, New York, N. Y.

BALLON, DAVID—1471 Crescent St., Montreal, Quebec
BAZIN, A. T.—1414 Drummond St., Montreal, Quebec
BERRY, FRANK B.—71 E. 71st St., New York, N. Y.
BETTMAN, RALPH B.—104 S. Michigan Ave., Chicago, Ill.
BIGGER, ISAAC A.—Medical College of Virginia, Richmond, Va.
BOLAND, FRANK K.—478 Peachtree St., N. E., Atlanta, Ga.
BUTLER, ETHAN FLAGG—956 West Water St., Elmira, N. Y.
BYERS, H. RODDICK—(Mail Returned) 3166 Westmont Blvd., Montreal, Quebec

CARLSON, HERBERT A.—4241 E. 14th St., Long Beach, Calif.
CLERF, LOUIS H.—1530 Locust St., Philadelphia, Pa.
COLE, DEAN B.—Professional Bldg., Richmond, Va.
CROWE, SAMUEL J.—Johns Hopkins Hospital, Baltimore, Md.

DAVISON, T. C.—478 Peachtree St., N. E., Atlanta, Ga.
DIEDERICH, VICTOR—236 Central Ave., Hot Springs, Ark.
DIEFFENBACH, RICHARD H.—570 Mt. Prospect Ave., Newark 4, N. J.
DOVELL, CHAUNCEY D., COL., M.C., U.S.A.—Regional Hospital, Fort Sheridan, Ill.

EGGERS, CARL—850 Park Ave., New York, N. Y.
EINHORN, MAX—20 E. 63rd St., New York, N. Y.
ELOESSER, LEO—490 Post St., San Francisco, Calif.

FAULKNER, WILLIAM B., JR.—1796 Geary St., San Francisco, Calif.
FERGUSON, R. G.—Fort San, Saskatchewan
FRANK, LOUIS WALLACELL—614 Heyburn Bldg., Louisville, Ky.

HARRINGTON, STUART W.—Mayo Clinic, Rochester, Minn.
HARVEY, SAMUEL C.—New Haven Hospital, New Haven, Conn.
HAYES, JOHN N.—24 Church St., Saranac Lake, N. Y.
HEINBECKER, PETER—Washington University Medical School, St. Louis, Mo.

JOHNS, FRANK S.—Johnston-Willis Hospital, Richmond, Va.

KERNAN, JOHN D.—103 E. 78th St., New York, N. Y.
KING, DONALD S.—Massachusetts General Hospital, Boston, Mass.

LAMBERT, ADRIAN V. S.—122 E. 76th St., New York, N. Y.
LEMON, WILLIS S.—510 10th Ave., S. W., Rochester, Minn.
LEWALD, LEON T.—1200 Fifth Ave., New York, N. Y.
LOCKWOOD, A. L.—300 Bloor St., E. Toronto, Ontario

MAES, URBAN—Pontchartrain Hotel, New Orleans, La.
MCSWEENEY, E. S.—(Mail Returned) 102 E. 35th St., New York, N. Y.
MILLER, ROBERT T., JR.—Mountain Lake, Lake Wales, Fla.
MYERS, J. ARTHUR—730 LaSalle Bldg., Minneapolis, Minn.

NEUHOF, HAROLD—1200 Fifth Ave., New York 29, N. Y.

ORNSTEIN, GEORGE—965 Fifth Ave., New York, N. Y.

PACKARD, EDWARD N.----142 Park Ave., Saranac Lake, N. Y.
PICKHARDT, OTTO C.----66 E. 79th St., New York, N. Y.

RIGLER, LEO G.----University Hospital, Minneapolis, Minn.

SHIPLEY, ARTHUR M.----University Hospital, Baltimore, Md.
SINGER, J. J.----616 N. Crescent Drive, Beverly Hills, Calif.
SMITH, DAVID T.----Duke University, Durham, N. C.
STETTEN, DEWITT----850 Park Ave., New York, N. Y.
STEWART, GEORGE A.----3301 N. Charles St., Baltimore, Md.

THEARLE, WILLIAM H.----221 W. Central Ave., Albuquerque, N. Mex.
THORBURN, GRANT----105 E. 53rd St., New York, N. Y.
TUCKER, GABRIEL----250 S. 19th St., Philadelphia, Pa.

VAN ALLEN, CHESTER M.----State Hospital, Bikaner, Rajputana, India

WHITTEMORE, WYMAN----199 Beacon St., Boston, Mass.

Members Deceased

Andrus, William De Witt
Brunn, Harold
Heuer, George J.
Lee, Walter Estell
Meyer, Alfred

INDEX TO VOLUME 21

A

Ackerman, Lauren (with Bosher, Lewis H., Jr., and Burford, Thomas H.) Pathology of experimentally produced lye burns and strictures of esophagus, 483

(with Jackson, Elisabeth, and Bertoli, Ferruccio) Exfoliative cytology—an adjunct in diagnosis of bronchiogenic carcinoma, 7

(with O'Neal, Lawrence W.) Cartilaginous tumors of ribs and sternum, 71

Ada, Alexander, E. W. (with Hevenor, Engel P.) Reconstruction of defects of thoracic wall with tantalum mesh gauze, 125

Adams, W. E. (with Carlson, Ralph F., Charbon, Bernard C., and Charbon, Harmia G. A.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621

Adenoma, bronchial—analysis of 26 cases (Delarue), 535

(Doty, Robert D.), 349

in supernumerary tracheal lobe, bronchial (Epstein), 362

Adhesions in opaque lobes, observations on division of (Coello), 135

American Association for Thoracic Surgery

List of members, 634

List of officers, council, membership committee, 633

Thirty-first annual meeting—program, 420

Anastomosis of blood vessels, clamps for end-to-end (Charbon), 109

Anesthetic explosions (Buckles), 426

Aneurysm with sternal perforation, successful repair of an aortic (Cowley, Sloan, and Sullenberger), 159

Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia (McCoy, Steinberg, and Dotter), 149

Angiocardiography, diagnostic value of dynamic studies in—evaluation of new rapid technique, (Keyes, Wegelius, and Lind), 164

Anomalous lobe of lung arising from esophagus (Gans and Potts), 313

origin of right subclavian artery from descending aorta, report of two cases of (Sealy), 319

Anomaly of pulmonary veins (Cooke, Evans, Kistin, and Blades), 452

Aorta at level of diaphragm treated successfully with preserved human blood vessel graft, coarctation of (Beattie, Cooke, Paul, and Orbison), 506

Aorta—Cont'd

in order to reach bifurcation of trachea, mobilization of arch of (Monod, Babou, Kateb, and Ghazi), 492

report of two cases of anomalous origin of right subclavian artery from descending (Sealy), 319

Artery from descending aorta, report of two cases of anomalous origin of right (Sealy), 319

lateral costal branch of internal mammary (Kropp), 421

Ashe, William M. (with McDonald, John R., and Clagett, O. Theron) Nonspecific pneumonitis of left upper lobe (simulating the "middle lobe syndrome" and producing early superior pulmonary sulcus syndrome), 1

Aspiration through endotracheal tube and extubation, sudden death occurring immediately after operation in patients with cardiac disease, with particular reference to role of (Shumacker and Hampton), 48

Atresia of esophagus treated by one-stage primary esophagogastrostomy employing right transpleural approach, 460

B

Babou, G. (with Monod, Olivier, Kateb, E., and Ghazi, S.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492

Beattie, Edward J., Jr. (with Cooke, Francis N., Paul, John S., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506

Beatty, Arch J. (with Buckingham, William W., and Ottosen, Poul), Resection Versus thoracoplasty in treatment of pulmonary tuberculosis, 34

(with Ottosen, Poul, Popp, Carol, and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202

Bertoli, Ferruccio (with Jackson, Elisabeth, and Ackerman, Lauren V.) Exfoliative cytology—an adjunct in diagnosis of bronchiogenic carcinoma, 7

Blades, Brian (with Cooke, Francis N., Evans, John M., and Kistin, Albert D.) Anomaly of pulmonary veins, 452

Blakemore, William S. (with Fowler, Ward S.) Lung function studies—VII. Effect of pneumonectomy on respiratory dead space, 433
Blalock, Alfred. Consideration of some of problems in cardiovascular surgery, 543
Blood vessels, clamps for end-to-end anastomosis of (Charbon), 109
volume and extracellular fluid in tuberculosis (Hay and Jenkins), 42
Bone, monostotic fibrous dysplasia of (Flickinger), 298
Bosher, Lewis H., Jr. Review of surgically treated lung abscess, 370
(with Burford, Thomas H., and Ackerman, Lauren) Pathology of experimentally produced lye burns and strictures of esophagus, 483
(with Taylor, Frederick H.) Heterotopic gastric mucosa in esophagus with ulceration and stricture formation, 306
Boyden, E. A. (with Hamre, C. J.) An analysis of variations in bronchovascular patterns of middle lobe in fifty dissected and twenty injected lungs, 172
Bradshaw, H. H. (with Myers, Richard T.) Benign intramural tumors and cysts of esophagus, 470
Brantigan, O. C. Oxygen administration: an emergency method when intratracheal tube fails with chest open, 532
Bronchial adenoma—analysis of 26 cases (Delarue), 535
in supernumerary tracheal lobe (Epstein), 362
nomenclature and present methods of treatment (Doty), 349
closure, effective method for (Rasmussen), 513
defects with stainless steel wire mesh, experimental reconstruction of tracheal and (Bucher and Burnett), 572
yardstick (Emerson), 516
Bronchiectasis, pulmonary artery in (Gobbel, Gordon, and Digman), 385
Bronchiogenic carcinoma, an adjunct in diagnosis of—exfoliative cytology (Jackson, Bertoli, and Ackerman), 7
neoplasm masquerading as alveolar cell carcinoma (Potts and Davidson), 402
Bronchoscopy in treatment of spontaneous and traumatic pneumothorax (Rubin and Rubin), 377
Bronchovascular patterns of middle lobe in fifty dissected and twenty injected lungs, an analysis of variations in (Boyden and Hamre), 172
Bucher, Robert M. (with Burnett, W. Emory) Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh, 572
Buckingham, William W. (with Beatty, Arch J., and Ottosen, Poul) Resection versus thoracoplasty in treatment of pulmonary tuberculosis, 34
Buckingham, William W.—Cont'd
(with Ottosen, Poul, Popp, Carol, and Beatty, Arch J.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202
Buckles, Maurice G. Anesthetic explosions, 426
Bullet embolus to left femoral artery following thoracic gunshot wound (Keeley), 608
Burford, Thomas H. (with Bosher, Lewis H., Jr., and Ackerman, Lauren) Pathology of experimentally produced lye burns and strictures of esophagus, 483
Burnett, W. Emory (with Bucher, Robert M.) Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh, 572

C

Carcinoma, bronchiogenic neoplasm masquerading as alveolar cell (Potts and Davidson), 402
of esophagus, paraganglioma, simulating (Taylor), 189
Cardiac disease, with particular reference to role of aspiration through endotracheal tube and extubation, sudden death occurring immediately after operation in patients with (Shumacker and Hampton), 48
Cardiovascular surgery, consideration of some of problems in (Blalock), 543
Carlson, Ralph F. (with Charbon, Bernard C., Charbon, Harmia G. A., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Carter, B. Noland (with Giuseffi, Jerome) Tracheotomy, useful procedure in thoracic surgery, with particular reference to employment in crushing injuries of thorax, 495
Cartilaginous tumors of ribs and sternum (O'Neal and Ackerman), 71
Charbon, Bernard C. Clamps for end-to-end anastomosis of blood vessels, 109
(with Carlson, Ralph F., Charbon, Harmia G. A., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Charbon, Harmia G. A. (with Carlson, Ralph F., Charbon, Bernard C., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Child, Charles G., III (with Harmon, Glen S., Dotter, Charles T., and Steinberg, Israel) Liver herniation simulating intrathoracic tumor, 391
Citret, Coleman (with Juvenelle, Andrea A., Wiles, Charles E., Jr., and Stewart, John D.) Pneumonectomy with replantation of lung in dog for physiologic study, 111

Clagett, O. Theron (with Ashe, William M., and McDonald, John R.) Nonspecific pneumonitis of left upper lobe (simulating "middle lobe syndrome" and producing early superior pulmonary sulcus syndrome), 1

(with Takaro, Timothy) Cavernous hemangioma of thoracic wall associated with clinical evidence of arteriovenous fistulas, 444

Clamps for end-to-end anastomosis of blood vessels (Charbon), 109

Clay, Richard C. (with Hanlon, C. Rollins) Pneumoperitoneum in differential diagnosis of diaphragmatic hernia, 57

Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft (Beattie, Cooke, Paul, and Orbison), 506

Coello, Alfred J. Observations on division of adhesions in opaque lobes, 135

Collapse therapy of pulmonary tuberculosis, importance of extrapleural pneumothorax in (Cutler), 217

Congenital absence of pericardium (Shafiroff), 30

Cooke, Francis N. (with Beattie, Edward J., Jr., Paul, John S., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506

(with Evans, John M., Kistin, Albert D., and Blades, Brian) Anomaly of pulmonary veins, 452

Cowley, R. Adams (with Sloan, Herbert) Posterior mediastinal tracheotomy, 602

(with Sloan, Herbert E., and Sullenberger, Neil H.) Successful repair of aortic aneurysm with sternal perforation, 159

Croxatto, Oscar C. (with Sampietro, Ruben) Pathology of pleural sclerosis: A study related to loss of expansivity of lungs and its treatment, 259

Cutler, J. W. Importance of extrapleural pneumothorax in collapse therapy of pulmonary tuberculosis, 217

Cysts of esophagus, benign intramural tumors and (Myers and Bradshaw), 470

D

Daniel, Rollin A., Jr. (with Diveley, Walter) Primary solitary neurogenic tumors of lung, 194

(with Sappington, Thomas B., Jr.) Accessory diaphragm, 212

Daume, Robert (with Eaton, Robert M., Sherwood, J. Vincent, Kendrick, Pearl L., and Eldering, Grace) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342

Davidson, Horace B. (with Potts, W. L.) Bronchiogenic neoplasm masquerading as alveolar cell carcinoma, 402

Delarue, N. C. Bronchial adenoma—analysis of 26 cases, 535

Diaphragm, accesory (Sappington, Thomas B., Jr., and Daniel, Rollin A., Jr.), 212
treated successfully with preserved human blood vessel graft, coarctation of aorta at level of (Beattie, Cooke, Paul, and Orbison), 506

Diaphragmatic hernia, pneumoperitoneum in differential diagnosis of (Clay and Hanlon), 57
with displaced liver, unusual (Knoepp), 394

Digman, George J. (with Gobbel, Walter G., Jr., and Gordon, Joseph) Pulmonary artery in bronchiectasis, 385

Diveley, Walter (with Daniel, Rollin A., Jr.) Primary solitary neurogenic tumors of lung, 194

Doppelt, Harry B. (with Goldberg, Jacob) Shoulder girdle dysfunction following thoracoplasty combined with partial scapulectomy, 584

Dotter, Charles T. (with Child, Charles G., III, Harmon, Glen S., and Steinberg, Israel) Liver herniation simulating intrathoracic tumor, 391

(with McCoy, Herbert I., and Steinberg, Israel) Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia, 149

Doty, Robert D. Bronchial adenoma—nomenclature and present methods of treatment, 349

Dynamic studies in angiocardiography, diagnostic value of—evaluation of new rapid technique (Keyes, Wegelius, and Lind), 164

E

Eaton, Robert M. (with Sherwood, J. Vincent, Kendrick, Pearl L., Eldering, Grace, and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342

Edwards, Jesse E. (with Peabody, Homer D., Jr., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, a pathologic study, 519

Eldering, Grace (with Eaton, Robert M., Sherwood, J. Vincent, Kendrick, Pearl L., and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342

Emerson, Ernest B., Jr. Bronchial yardstick, 516

Emphysema with defect of anterior mediastinum, obstructive (Lewis and Potts), 438

Empyema, use of streptokinase-streptodornase in treatment of chronic (Tillett, Sherry, and Read), 325
of postpneumonic (Tillett, Sherry, and Read), 275

Endotracheal tube and extubation, sudden death occurring immediately after operation in patients with cardiac disease, with particular reference to role of aspiration through (Shumacker and Hampton), 48

Eosinophilic granuloma of rib (Kipp and Fisher), 24

Epstein, Isaac. Bronchial adenoma in supernumerary tracheal lobe, 362

Esophagogastrostomy employing right transpleural approach, congenital atresia of esophagus treated by one-stage primary (Madden), 460

Esophagostomy, cervical (Klopp), 490

Esophagus, anomalous lobe of lung arising from (Gans and Potts), 313
benign intramural tumors and cysts of (Myers and Bradshaw), 470
paraganglioma simulating carcinoma of (Taylor), 189
pathology of experimentally produced lye burns and strictures of (Bosher, Burford, and Ackerman), 483
treated by one-stage primary esophagogastrostomy employing right transpleural approach (Madden), 460
with ulceration and stricture formation, heterotopic gastric mucosa in (Bosher and Taylor), 306

Evans, John M. (with Cooke, Francis N., Kistin, Albert D., and Blades, Brian) Anomaly of pulmonary veins, 452

Exfoliative cytology—an adjunct in diagnosis of bronchiogenic carcinoma (Jackson, Bertoli, and Ackerman), 7

F

Femoral artery following a thoracic gunshot wound, bullet embolus to left (Keeley), 608

Fibrosis, clinically indeterminate pulmonary —pathologic study (Peabody, Moersch, and Edwards), 519

Fisher, Edwin R. (with Kipp, Harold A). Eosinophilic granuloma of rib, 24

Fistulas, cavernous hemangioma of thoracic wall associated with clinical evidence of arteriovenous (Takaro and Clagett), 444

Flickinger, F. Miles. Monostotic fibrous dysplasia of bone—report of case involving three contiguous ribs treated by wide resection of thoracic cage, 298

Fowler, Ward S. (with Blakemore, William S.) Lung function studies—VII. Effect of pneumonectomy on respiratory dead space, 433

G

Gans, Stephen L. (with Potts, Willis J.) Anomalous lobe of lung arising from esophagus, 313

Ghazi, S. (with Monod, Olivier, Babou, G., and Kateb, E.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492

Giuseffi, Jerome (with Carter, B. Noland) Tracheotomy, useful procedure in thoracic surgery, with particular reference to employment in crushing injuries of thorax, 495

Gobbel, Walter G., Jr. (with Gordon, Joseph, and Digman, George J.) Pulmonary artery in bronchiectasis, 385

Goldberg, Jacob (with Doppelt, Harry B.) Shoulder girdle dysfunction following thoracoplasty combined with partial scapulectomy, 584

Gordon, Joseph (with Gobbel, Walter G., Jr., and Digman, George J.) Pulmonary artery in bronchiectasis, 385

Graft, coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel (Beattie, Cooke, Paul, and Orbison), 506

Granuloma of rib, eosinophilic (Kipp and Fisher), 24

H

Hamre, C. J. (with Boyden, E. A.) An analysis of variations in bronchovascular patterns of middle lobe in fifty dissected and twenty injected lungs, 172

Hanlon, C. Rollins (with Clay, Richard C.) Pneumoperitoneum in differential diagnosis of diaphragmatic hernia, 57

Harmon, Glen S. (with Child, Charles G., III, Dotter, Charles T., and Steinberg, Israel) Liver herniation simulating intrathoracic tumor, 391

Hay, Celestine R. (with Hay, Elliott B., and Jenkins, Daniel E.) Blood volume and extracellular fluid in tuberculosis, 42

Hay, Ellott B. (with Hay, Celestine R., and Jenkins, Daniel E.) Blood volume and extracellular fluid in tuberculosis, 42

Hellsten, Helge (with Ramstrom, Sven) Surgical treatment of three cases of pleural sarcoma, 116

Hemangioma of thoracic wall associated with clinical evidence of arteriovenous fistulas, cavernous (Takaro, Timothy, and Clagett, O. Theron), 444

Hemopneumothorax, pulmonary decortication for clotted hemothorax following idiopathic spontaneous (Seley and Neuhof), 600

Hemothorax following idiopathic spontaneous hemopneumothorax, pulmonary decortication for clotted (Seley and Neuhof), 600

Heterotopic gastric mucosa in esophagus with ulceration and stricture formation (Bosher and Taylor), 306

Hevenor, Engel P. (with Ada, Alexander, E. W.) Reconstruction of defects of thoracic wall with tantalum mesh gauze, 125

Homotransplantation of tracheal segments preserved by lyophilization—experimental study (Marrangoni), 398

I

Intratracheal tube fails with chest open, emergency method when—oxygen administration (Brantigan), 532

J

Jackson, Elisabeth (with Bertoli, Ferruccio, and Ackerman, Lauren V.) Exfoliative cytology—adjunct in diagnosis of bronchiogenic carcinoma, 7

Jenkins, Daniel E. (with Hay, Elliott B., and Hay, Celestine R.) Blood volume and extracellular fluid in tuberculosis, 42

Jennings, Hampton L. (with Shumacker, Harris B., Jr.) Sudden death occurring immediately after operation in patients with cardiac disease, with particular reference to role of aspiration through endotracheal tube and extubation, 48

Juvenelle, Andre A. (with Citret, Coleman, Wiles, Charles E., Jr., and Stewart, John D.) Pneumonectomy with replantation of lung in dog for physiologic study, 111

K

Kateb, E. (with Monod, Olivier, Babou, G., and Ghazi, S.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492

Keeley, John L. Bullet embolus to left femoral artery following thoracic gunshot wound, 608

Kendrick, Pearl L. (with Eaton, Robert M., Sherwood, J. Vincent, Eldering, Grace, and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342

Keyes, T. F. (with Wegelius, C., and Lind, J.) The diagnostic value of dynamic studies in angiocardiography, evaluation of new rapid technique, 164

Kipp, Harold A. (with Fisher, Edwin R.) Eosinophilic granuloma of rib, 24

Kistin, Albert D. (with Cooke, Francis N., Evans, John M., and Blades, Brian) Anomaly of pulmonary veins, 452

Klopp, Calvin T. Cervical esophagostomy, 490

Knoepp, Louis F. Unusual diaphragmatic hernia with displaced liver, 394

Kropp, Benjamin N. Lateral costal branch of internal mammary artery, 421

L

Lewis, James E. (with Potts, Willis J.) Obstructive emphysema with defect of anterior mediastinum, 438

Lind, J. (with Keyes, T. F., and Wegelius, C.) The diagnostic value of dynamic studies in angiocardiography, evaluation of new rapid technique, 164

Liver herniation simulating intrathoracic tumor (Child, Harmon, Dotter, and Steinberg), 391

unusual diaphragmatic hernia with displaced (Knoepp), 394

Lobe, bronchial adenoma in supernumerary tracheal (Epstein), 362

Lung abscess, review of surgically treated (Bosher), 370

function studies. VII. Effect of pneumonectomy on respiratory dead space (Fowler and Blakemore), 433

in dog for physiologic study, pneumonectomy with replantation of (Juvenelle, Citret, Wiles, and Stewart), 111

primary solitary neurogenic tumors of (Diveley and Daniel), 194

tissue on right ventricular pressures in animals, effect of decreasing amount of (Carlson, Charbon, Charbon, and Adams), 621

Lungs, analysis of variations in bronchovascular patterns of middle lobe in fifty dissected and twenty injected (Boyden and Hamre), 172

and its treatment, study related to loss of expansivity of—pathology of pleural sclerosis (Croxatto and Sampietro), 259

Lye burns and strictures of esophagus, pathology of experimentally produced (Bosher, Burford, and Ackerman), 483

Lyophilization, homotransplantation of tracheal segments preserved by—experimental study (Marrangoni), 398

M

Madden, John L. Congenital atresia of esophagus treated by one-stage primary esophagogastrostomy employing right transpleural approach, 460

Marrangoni, Albert G. Homotransplantation of tracheal segments preserved by lyophilization; experimental study, 398

McCoy, Herbert I. (with Steinberg, Israel, and Dotter, Charles T.) Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia, 149

McDonald, John R. (with Ashe, William M., and Clagett, O. Theron) Nonspecific pneumonitis of left upper lobe (simulating ''middle lobe syndrome'' and producing early superior pulmonary sulcus syndrome), 1

Mediastinal tracheotomy, posterior (Adams and Sloan), 602

Mediastinum, obstructive emphysema with defect of anterior (Lewis and Potts), 438

Middle lobe in fifty dissected and twenty injected lungs, analysis of variations in bronchovascular patterns of (Boyden and Hamre), 172

''Middle lobe syndrome'' and producing early superior pulmonary sulcus syndrome, simulating nonspecific pneumonitis of left upper lobe (Ashe, McDonald, and Clagett), 1

Moersch, Herman J. (with Peabody, Homer D., Jr., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, pathologic study, 519

Monod, Olivier (with Babou, G., Kateb, E., and Ghazi, S.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492

Monostotic fibrous dysplasia of bone (Flickinger), 298

Myers, Richard T. (with Bradshaw, H. H.) Benign intramural tumors and cysts of esophagus, 470

N

Neuhof, Harold (with Seley, Gabriel P.) Pulmonary decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax, 600

O

O'Neal, Lawrence W. (with Ackerman, Lauren V.) Cartilaginous tumors of ribs and sternum, 71

Opaque lobes, observations on division of adhesions in (Coello), 135

Orbison, James A. (with Beattie, Edward J., Jr., Cooke, Francis N., and Paul, John S.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506

Osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, serum protein levels and calculated (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342

Ottosen, Poul (with Beatty, Arch J., and Buckingham, William W.) Resection versus thoracoplasty in treatment of pulmonary tuberculosis, 34

(with Popp, Carol, Beatty, Arch J., and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202

Oxygen administration: emergency method when intratracheal tube fails with chest open (Brantigan), 532

P

Paraganglioma simulating carcinoma of esophagus (Taylor), 189

Paul, John S. (with Beattie, Edward J., Jr., Cooke, Francis N., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506

Peabody, Homer D., Jr. (with Moersch, Herman J., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, pathologic study, 519

Pericardium, congenital absence of (Shafiroff), 30

Phreniclasia, angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and (McCoy, Steinberg, and Dotter), 149

Pleural sarcoma, surgical treatment of three cases of (Ramstrom and Hellsten), 116

sclerosis, pathology of—study related to loss of explansivity of lungs and its treatment (Croxatto and Sampetro), 259

Pneumonectomy on respiratory dead space, effect of lung function studies (Fowler and Blakemore), 433

with replantation of lung in dog for physiologic study (Juvenelle, Citret, Wiles, and Stewart), 111

Pneumonitis of left upper lobe (simulating ''middle lobe syndrome'' and producing early superior pulmonary sulcus syndrome), nonspecific (Ashe, McDonald, and Clagett), 1

Pneumoperitoneum, and phreniclasia, angiocardiographic findings in thoracoplasty, artificial (McCoy, Steinberg, and Dotter), 149

in differential diagnosis of diaphragmatic hernia (Clay and Hanlon), 57

Pneumothorax, bronchoscopy in treatment of spontaneous and traumatic (Rubin and Rubin), 377

in collapse therapy of pulmonary tuberculosis, importance of extrapleural (Cutler), 217

Popp, Carol (with Ottosen, Poul, Beatty, Arch J., and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202

Potts, Willis J. (with Gans, Stephen L.) Anomalous lobe of lung arising from the esophagus, 313

(with Lewis, James E.) Obstructive emphysema with defect of anterior mediastinum, 438

Potts, W. L. (with Davidson, Horace B.) Bronchiogenic neoplasm masquerading as alveolar cell carcinoma, 402

Presidential address (Blalock), 543
Pulmonary artery in bronchiectasis (Gobbel, Gordon, and Digman), 385
decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax (Seley and Neuhof), 600
fibrosis, clinically indeterminate—pathologic study (Peabody, Moersch, and Edwards), 519
sulcus syndrome, nonspecific pneumonitis of left upper lobe simulating "middle lobe syndrome" and producing early superior (Ashe, McDonald, and Clagett), 1
tuberculosis, evaluation of results in different types of—one hundred thirty consecutive thoracoplasties (Ottosen, Bopp, Beatty, and Buckingham), 202
importance of extrapleural pneumothorax in collapse therapy of (Cutler), 217
resection versus thoracoplasty in treatment of (Beatty, Buckingham, and Ottosen), 34
veins, anomaly of (Cooke, Evans, Kistin, and Blades), 452

R

Ramstrom, Sven (with Hellsten, Helge) Surgical treatment of three cases of pleural sarcoma, 116
Rasmussen, Richard A. Effective method for bronchial closure, 513
Read, C. Thomas (with Tillett, Wm. S., and Sherry, Sol) Use of streptokinase-streptodornase in treatment of chronic empyema with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325
(with Tillett, William S., and Sherry, Sol) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
Resection versus thoracoplasty in treatment of pulmonary tuberculosis (Beatty, Buckingham, and Ottosen), 34
Rib, eosinophilic granuloma of (Kipp and Fisher), 24
Ribs and sternum, cartilaginous tumors of (O'Neal and Ackerman), 71
Rubin, Eli H. (with Rubin, Morris) Bronchoscopy in treatment of spontaneous and traumatic pneumothorax, 377
Rubin, Morris (with Rubin, Eli H.) Bronchoscopy in treatment of spontaneous and traumatic pneumothorax, 377

S

Sampietro, Ruben (with Croxatto, Oscar C.) Pathology of pleural sclerosis: Study related to loss of expansivity of the lungs and its treatment, 259
Sappington, Thomas B., Jr. (with Daniel, Rollin A, Jr.) Accessory diaphragm, 212
Sarcoma, surgical treatment of three cases of (Ramstrom and Hellsten), 116
Scapulectomy, shoulder girdle dysfunction following thoracoplasty combined with partial (Doppelt and Goldberg), 584
Sealy, W. C., Report of two cases of anomalous origin of right subclavian artery from descending aorta, 319
Seley, Gabriel P. (with Neuhof, Harold) Pulmonary decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax, 600
Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342
Shafiroff, B. G. P. Congenital absence of pericardium, 30
Sherry, Sol (with Tillett, William S. and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of chronic empyema—with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325
(with Tillett, William S., and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
Sherwood, J. Vincent (with Eaton, Robert M., Kendrick, Pearl L., Eldering, Grace, and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342
Shoulder girdle dysfunction following thoracoplasty combined with partial scapulectomy (Doppelt and Goldberg), 584
Shumacker, Harris B., Jr. (with Hampton, L. Jennings) Sudden death occuring immediately after operation in patients with cardiac disease, with particular reference to role of aspiration through endotracheal tube and extubation, 48
Sloan, Herbert E. (with Cowley, R. Adams) Posterior mediastinal tracheotomy, 602
(with Cowley, R. Adams, and Sullenberger, Neil H.) Successful repair of aortic aneurysm witih sternal perforation, 159
Steinberg, Israel (with Child, Charles G., III, Harmon, Glen S., and Dotter, Charles T.) Liver herniation simulating intrathoracic tumor, 391
(with McCoy, Herbert I., and Dotter, Charles T.) Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia, 149
Sternal perforation, successful repair of aortic aneurysm with (Cowley, Sloan, and Sullenberger), 159

Stewart, John D. (with Juvenelle, Andre A., Citret, Coleman, and Wiles, Charles E., Jr.) Pneumonectomy with replantation of lung in dog for physiologic study, 111
Streptokinase-streptodornase in treatment of chronic empyema, use of (Tillett, Sherry, and Read), 325
postpneumonic empyema (Tillett, Sherry, and Read), 275
Stricture formation, heterotopic gastric mucosa in esophagus with ulceration and (Bosher and Taylor), 306
Sullenberger, Neil H. (with Cowley, R. Adams, and Sloan, Herbert E.) Successful repair of aortic aneurysm with sternal perforation, 159

T

Takaro, Timothy (with Clagett, O. Theron) Cavernous hemangioma of thoracic wall associated with clinical evidence of arteriovenous fistulas, 444
Tantalum mesh gauze, reconstruction of defects of thoracic wall with (Ada and Hevenor), 125
Taylor, Frederick H. Paraganglioma simulating carcinoma of esophagus, 189
(with Bosher, Lewis H., Jr.) Heterotopic gastric mucosa in esophagus with ulceration and stricture formation, 306
Thoracic gunshot wound, bullet embolus to left femoral artery following (Keeley), 608
wall associated with clinical evidence of arteriovenous fistulas, cavernous hemangioma of (Takaro and Clagett), 444
with tantalum mesh gauze, reconstruction of defects (Ada and Hevenor), 125
Thoracoplasties, one hundred thirty consecutive—evaluation of results in different types of pulmonary tuberculosis (Ottosen, Bopp, Beatty, and Buckingham), 202
Thoracoplasty, artificial pneumoperitoneum, and phreniclasia, angiocardiographic findings in (McCoy, Steinberg, and Dotter), 149
combined with partial scapulectomy, shoulder girdle dysfunction following (Doppelt and Goldberg), 584
in treatment of pulmonary tuberculosis, resection versus (Beatty, Buckingham, and Ottosen), 34
Thorax, tracheotomy, useful procedure in thoracic surgery, with particular reference to its employment in crushing injuries of (Carter and Giuseffi), 495
Tillett, William S. (with Sherry, Sol, and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
(with Sherry, Sol, and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of chronic empyema—with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325
Trachea, mobilization of arch of aorta in order to reach bifurcation of (Monod, Babou, Kateb, and Ghazi), 492
Tracheal and bronchial defects with stainless steel, experimental reconstruction of (Bucher and Burnett), 572
segments preserved by lyophilization, homotransplantation of—experimental study (Marrangoni), 398
Tracheotomy, posterior mediastinal (Sloan and Cowley), 602
useful procedure in thoracic surgery, with particular reference to its employment in crushing injuries of thorax (Carter and Giuseffi), 495
Tuberculosis, blood volume and extracellular fluid in (Hay and Jenkins), 42
serum protein levels and calculated osmotic pressure in: I. Correlation of values with severity of disease (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342
Tumor, liver herniation simulating intrathoracic (Child, Harmon, Dotter, and Steinberg), 391
Tumors and cysts of esophagus, benign intramural (Myers and Bradshaw), 470
of lung, primary solitary neurogenic (Diveley and Daniel), 194

V

Ventricular pressures in animals, effect of decreasing amount of lung tissue on right (Carlson, Charbon, Charbon, and Adams), 621

W

Wegelius, C. (with Keyes, T. F., and Lind, J.) Diagnostic value of dynamic studies in angiocardiography, evaluation of new rapid technique, 164
Wiles, Charles E., Jr. (with Juvenelle, Andre A., Citret, Coleman, and Stewart, John D.) Pneumonectomy with replantation of lung in dog for physiologic study, 111